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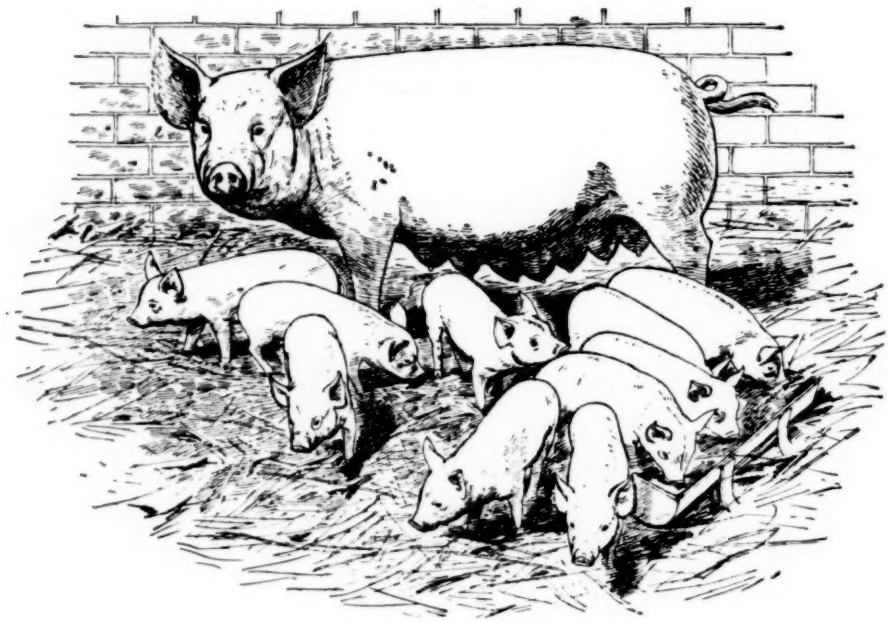

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Deaths in Pigs on a High Copper Diet

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SUMMARY.—Serious losses in fattening pigs associated with the feeding of meal to which a copper supplement had been added are described and discussed in relation to recorded cases of chronic copper poisoning. It is suggested that the dangerously high copper levels found in the liver and kidneys of all pigs examined together with some other unidentified toxic factor in the diet may have caused the losses.

Introduction

THE literature regarding toxicity of copper salts for domestic animals was well reviewed by Boughton and Hardy (1934) and by Eden (1940). The former reported that in the latter part of the past century Ellenberger and Hofmeister (1883) and Baum and Seeliger (1898) proved definitely that chronic copper poisoning, following the continued ingestion of small amounts of various copper salts, is a distinct clinical entity in domestic animals. Eden pointed out that a search of the literature showed that there was a considerable diversity of opinion as to whether small quantities of copper ingested over a considerable period of time could exert toxic effects. However, the results of the investigations of Boughton and Hardy (1934) and of Eden (1940) leave no doubt that toxic effects can be caused in sheep by the continued ingestion of small amounts of copper sulphate. In general, sheep appear to accumulate copper more readily than do other species under similar conditions of high copper intake (Underwood, 1956). Most of the more recently recorded cases of copper poisoning have been in sheep (Fincham, 1945; Ogilvie, 1954; Clegg, 1956; Pearson, 1956) associated with grazing in orchards sprayed with copper fungicides, or with the use of dietary copper supplements which are frequently given irrespective of whether a low copper status is present or not in the particular animals. Although the use of some form of copper supplement for sheep and cattle has been a fairly common practice among farmers in this country over the last 10 years, it is only recently that its use has been extended to pigs. This followed the report by Barber, Braude and Mitchell (1955), which indicated that addition of copper at the rate of 250 p.p.m. to the ration of fattening pigs enhanced the growth rate.

A search of the literature suggests that copper poisoning in pigs is not common. This is probably because, until the recent use of copperised pig meals, they have not been exposed to the risk of overdosage with prophylactic or therapeutic copper compounds to the same extent as sheep and cattle. A recent outbreak has, however, been reported by Gordon and Luke (1957) in which it was known that the meal had been supplemented with copper sulphate although it was not possible to ascertain the amount of copper which had been fed.

In the present report, an account is given of deaths in pigs associated with the feeding of meal containing a mineral mixture which included copper sulphate as one of the ingredients. The case involved the loss of 23 fattening pigs in a period of some 5 to 6 weeks and the condemnation of a number of carcases of pigs slaughtered at bacon weight. Although it is felt that assessment of the part played by copper in this case must await a description of the lesions of experimental chronic copper poisoning in the pig, the fact that it was found that pigs, in common with sheep and calves, can absorb and store dangerous amounts of copper made it seem desirable to record the details and point out the possible risk associated with the feeding of copper-supplemented meals to pigs.

Clinical History

The herd consisted of approximately 200 pigs (Large White and Large White × Landrace). The first death which might be attributed to this cause occurred in November, 1955. This was a store pig approximately 4 months' old which died suddenly, and *post-mortem* examination had shown an extensive recent haemorrhage into the stomach, blood-stained intestinal contents and a pale cirrhotic liver. In the following month a batch of 51 pigs had been sent for slaughter and of these, 4 carcases were totally condemned because of jaundice and three others

partially condemned for the same reason. No further deaths occurred until mid-January, 1956, but from this time until the middle of February, deaths occurred at the rate of 3 to 4 pigs a week, until a total of 23 pigs aged between 3 and 7 months had died. In addition, out of 42 pigs slaughtered at bacon weight, 4 carcases were totally condemned and one partially condemned as jaundiced.

In the fatal cases, illness was of sudden onset and short duration, all pigs being "found dead" with the exception of the last one to die. This animal was noticed to be slightly dull late one night but was not thought to be seriously ill. It was, however, found dead early on the following morning.

Examination of the pigs on the farm during the second week in February showed a number of recently weaned young stores and also a few older fattening pigs to be affected with a dry scurfy condition ("dry-feeding dermatitis"); many pigs were coughing, there was some variation in size of pigs in a few pens and one jaundiced pig was seen. This last animal was in good condition and showed no abnormality apart from its slightly jaundiced appearance. The great majority of the pigs were, however, in good thriving condition and showed no signs of ill health. The deaths had occurred in various pens both on the home premises and on a second farm where pigs were also kept under similar conditions of feeding and management but with separate attendants. The surviving pigs in the pens in which the deaths had occurred were all apparently healthy. Some rats were present on the farm, though not in large numbers, and no attempt had been made to destroy them apart from sporadic trapping.

In view of the history of the losses and the *post-mortem* picture, poisoning was suspected as being the cause of death. Enquiries on the farm indicated that from the time of weaning until removed for slaughter, the pigs were confined to pens and had no access to food other than that fed to them; they were bedded in clean straw and water was drawn direct from the public supply. There was no evidence of access to or availability of, any toxic agent apart from the possibility of some such material having been incorporated in the meal which was being fed, or of deliberate malicious poisoning. Chemical analyses were therefore carried out on the meal as fed to the pigs, on the purchased concentrate mixture and on samples of stomach contents and various tissues from dead pigs. In the meantime a change of feeding was advised and the owner decided to use a purchased proprietary pig meal. In the first 6 days following the change of diet, 4 pigs died but after that time no similar losses occurred in a period of 7 months following the change of feeding, although a few carcases of pigs slaughtered at bacon weight were partially condemned because of jaundice.

Feeding and Management.

Young litters were creep-fed with pellets until weaned at approximately 8 weeks of age, after which the piglets were dry-fed with a meal mixture prepared along lines suggested by the National Agricultural Advisory Service to enable the farmer to utilise his home-grown cereals. These cereals were ground on the farm, then mixed through a mechanical mixer with a concentrate mixture obtained ready-mixed from a local mill. This concentrate consisted of fish meal, ground nut meal, (or soya bean meal), meat meal, vitamins, antibiotic and minerals, and was mixed with the home-grown cereals in the proportion of 1½ cwt. to 8 cwt cereals (barley, oats and wheatings).

On the farm, the home-grown cereals and the purchased concentrate were stored in the barn loft. They were emptied into the mixing machine in the loft and the complete meal mixture removed from the machine at ground level and stored there. The farmer was always present at the time the meal was mixed and there appears to be no likelihood of a mistake having been made in the composition of the meal mixture. However, the mixing machine had been newly installed on the farm in December, 1955, and home mixing done for the first time on December 16th. Before this date exactly the same meal mixture was fed to the pigs but mixing was done wholly by the local miller.

The complete meal mixture was fed *ad lib.* dry in hoppers from weaning to approximately 100 lb. live-weight. At this stage the pigs were transferred to the fattening house where the same meal was fed, again in the dry state but rationed to 3½ lb. meal per pig per day; the amount of meal fed was increased gradually during the fattening period to a maximum of 5½ lb. per pig per day for pigs over 180 lb. live-weight. No supplementary food was given and a separate supply of water was always available, being obtained direct from the main public water supply.

Litters were reared outside on pasture, housed in wooden arks until weaned. They were then transferred to brick and concrete built pens with no outside run and between weaning and slaughter the pigs were managed wholly on the intensive system.

Results of *Post-mortem*, Pathological and Chemical Examinations (a) Post-mortem *Findings.*

The carcases of 11 pigs which died during the course of the outbreak were examined at the laboratory. The main findings were pale and anaemic carcases with varying degrees of liver and kidney damage and jaundice, together with extensive haemorrhage into the stomach or large intestine and/or subcutaneously into one or more of the legs. In most cases, but not in all, loss of blood appeared to be the immediate cause of death.

The findings varied somewhat between carcases but those seen in the first 3 pigs and in the last pig examined were fairly typical of the whole and are given in some detail:—

1. Gilt: found dead. The carcase was pale and anaemic in appearance but showed no other external lesions. The stomach was full of dark straw bedding, the small intestine was pale and slightly distended with gas while in the caecum and colon the mucosa was diffusely congested and contained dark blood-stained ingesta. The liver was cirrhotic and markedly yellow throughout its substance. The spleen was thought to be slightly enlarged and the kidneys were

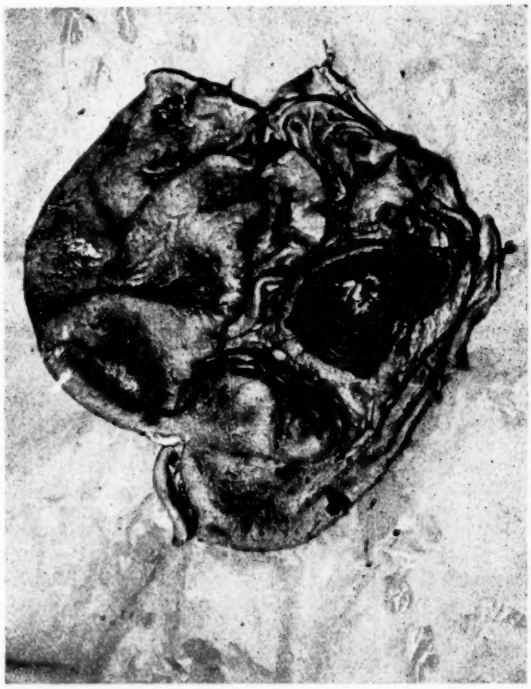
"bronzed" and had a metallic lustre; a little white, turbid urine was present in the bladder. The lungs were slightly oedematous and yellow while the heart was pale. The carcase had a jaundiced appearance due to yellow discoloration of the body fat.

2. Male (castrated): found dead. The stomach was full of clotted blood which had apparently originated from a large penetrating ulcer which surrounded the oesophageal opening. The remainder of the gastric mucous membrane was normal in appearance. Much blood was present in the small intestine but traces only were seen in the large gut. The liver was a pale rose-pink colour and was firm and cirrhotic. The spleen and kidneys were pale and the lungs showed localised areas of pneumonia in the apical lobes only. Jaundice was not obvious in this carcase.

3. Male (castrated): found dead. The carcase was pale and anaemic and extensive subcutaneous haemorrhages were present in both hind legs and the right fore leg. The stomach was again full of clotted blood and a large ulcer, approximately two inches in diameter, surrounded the oesophageal entrance. The contents of the small and large intestines were blood-stained. The liver and kidneys were bronzed in appearance and there was yellow discoloration of the body fat. Small localised areas of pneumonia were found in the lungs. The regional lymphatic glands associated with the legs into which haemorrhage had occurred were stained with blood.

4. Gilt: slightly dull late one night and found dead early on the following morning. The skin was slightly jaundiced, traces of blood were seen around the anus and the left hind leg showed extensive purple discoloration and swelling due to the presence of a large subcutaneous haemorrhage. The stomach and small intestine contained normal food material and showed no evidence of ulceration or haemorrhage; the contents of the large intestine were, however, blood-stained and contained some blood clots. The liver was firm and rather fibrous and was markedly yellow throughout its substance. The kidneys, lungs and body fat were slightly jaundiced. The lymphatic glands associated with the left hind leg contained blood.

In the 11 carcases examined evidence of jaundice was found in 8 cases, and in 5 of these pigs yellow discoloration of the skin was observed before the carcase was opened. Ulceration of the stomach was noted in 7 carcases; in one of these no haemorrhage had occurred into the stomach but in the other 6 instances severe haemorrhage had occurred. Three of the pigs examined showed haemorrhage into the lumen of the large intestine when no evidence of ulceration or of bleeding was found higher up the alimentary canal. In 3 carcases, there were extensive subcutaneous haemorrhages in the legs, causing swelling and purple discoloration of the skin; 1 leg was affected in 1 pig, 3 in the second and all 4 legs in the third case. In 1 pig only, no evidence of haemorrhage was found at *post-mortem* examination; ulceration around the oesophageal opening into the stomach was present in this carcase and the liver was rose-pink in colour and rather fibrous. Cirrhosis of the liver was noted in 5 carcases and in a few more the liver appeared to be rather more firm and fibrous than is normal. Generally, the more severe the liver damage, the more extensive and intense the jaundice which was present.



Pig stomach: showing area of ulceration surrounding entrance of oesophagus.

The livers of 2 pigs which were condemned at slaughter were examined. Each was very firm in consistency, cirrhotic and markedly yellow throughout the substance; one had a typical "hob-nailed" appearance and showed small areas of haemorrhage in the substance.

(b) *Pathological Examinations.*

Extensive cultural examinations of the viscera and muscles were carried out under aerobic and anaerobic conditions with completely negative results except for the isolation of a *Pasteurella*-like organism from the pneumonic patches of lung tissue in 3 pigs. This infection appeared to be quite localised and was recovered only from consolidated lung tissue.

Microscopical examination of urine under dark ground illumination and biological tests, carried out by the intraperitoneal inoculation of young guinea-pigs with urine and suspensions of liver and kidney from dead pigs, proved negative for evidence of leptospirosis. Histological examination of liver and kidneys also failed to show the presence of *Leptospira*. Samples of blood plasma from 5 dead pigs and of blood serum from 9 other pigs on the farm were examined for leptospira agglutinins against suspen-

sions of *L. canicola, L. icterohaemorrhagiae, L. pomona, L. hyos* and *L. grippo-typhosa*. Two samples only (from live pigs on the farm, neither of which died) gave titres in excess of 1:10, in each case against *L. icterohaemorrhagiae*. Repeat blood samples were obtained from each of these animals one week later when the pig which had shown a titre of 1:100 at the first examination gave a negative reaction and the second animal remained constant at a titre of 1:30. One rat, caught alive on the farm during the period in which losses were occurring, proved negative for evidence of *Leptospira* infection.

Histological examination was carried out on livers from 7 pigs, kidneys from 4 pigs, and on stomachs from 4 pigs by the Pathology Department of the Ministry's Central Veterinary Laboratory and the following is a summary of the lesions which were found.

Liver: The degree of abnormality in the 7 livers examined varied considerably and they fell roughly into 3 groups.

The first group displayed the least abnormality and comprised the livers of 2 pigs which died apparently of haemorrhage from ulcers of the stomach. These showed a widespread mild centrilobular degeneration. The parenchymal cells of the degenerate areas were irregular in size and shape; some contained numerous, rather indistinct vacuoles, while others showed such necrobiotic changes as margination and bleeding of nuclear chromatin, karyorrhexis and karyolysis. A number of Kupffer cells and parenchymal cells contained faintly brown granules, and there were scattered intracellular and extracellular deposits of bile. Some affected areas showed slight infiltration with lymphocytes and polymorphs. The normal pattern of sinusoids and cell cords was preserved. In some cases there was slight thickening of the septa and early formation of intercellular collagen in the degenerate areas.

The second group included the livers from 1 pig condemned for jaundice at the abattoir, 1 pig which died with haemorrhage from ulceration of the stomach, and 2 pigs which showed no ulceration or haemorrhage into the stomach but in which haemorrhage had occurred into the large intestine and, in 1 animal, subcutaneously in one leg. In this group the septal fibrosis was greater. The lobules appeared distorted and enlarged; some retained a recognisable architecture, with centrilobular congestion and degeneration, but a proportion of intact sinusoids; others had lost their arrangement into cords and were made up of scattered widely separated groups of cells, irregular and varying in size. The space between the cell groups contained no recognisable sinusoids; but was filled by fine strands of collagen and an amorphous, eosinophilic, fibrin-like debris. Intracellular and extracellular masses of bile were more numerous. The hepatic cells were more heavily vacuolated, some contained faintly brown granules, others showed round, intracytoplasmic eosinophilic inclusions of varying size, which stained orange-brown by Masson's trichrome method. Within the new fibrous tissue there was some proliferation of bile ducts, but this was not a marked feature.

Lymphocytes and polymorphs were present in moderate concentration in the areas of centrilobular degeneration and also formed small foci scattered through the septa.

The third group was characterised by the "hob-nailed" liver from a pig condemned because of jaundice when sent for slaughter. The liver lobules were still recognisable by virtue of the greater density of the septal fibrous tissue, but appeared grossly enlarged. A few lobules still consisted mainly of parenchymal cells, which were irregular in shape and size, without formation of regular cords. Several of these "normal" lobules had in addition, undergone acute haemorrhagic necrosis, with considerable lymphocytic and polymorph infiltration. The majority of the macroscopically outlined lobules were made up of irregular, degerenate parenchymal cells, scattered singly and in small groups through a groundwork of fibrous tissue. Proliferating bile ducts were present, but not strikingly numerous; bile masses were common.

There was little evidence of regeneration in most of these livers other than a few round nodules of parenchymal cells, varying in size and shape and showing occasional giant nuclei. Some showed areas of eosinophile infiltration of the septa, which were believed to be due to concurrent hepatitis eosinophilica. When stained by the rubeanic acid method of Okamoto and Utamura, the less damaged livers showed olive-green granules. These were numerous and intensely stained in the Kupffer cells but less numerous and paler in the parenchymal cells. No such material could be demonstrated in the more severely damaged livers. The most severely damaged livers showed some resemblance to hepatosis diaetetica acuta et chronica, but the overall picture seemed to indicate a completely different course of development. Despite the numerous bile thrombi, the type and distribution of the lesions did not suggest a primarily obstructive cholangio-hepatitis.

Stomach: In all cases the ulceration was confined to the oesophageal zone of the stomach. There was complete necrosis of the epithelium, under which was a layer of fibrous tissue which varied in depth. This usually penetrated into, and mingled with the muscular coats, and under-ran the muscularis mucosae of the intact, glandular epithelium for a short distance. This fibrous tissue and the adjacent submucosa showed scattered areas of oedema. Leucocytic reaction was generally moderate and consisted of a zone of very degenerate cells in the border of the necrotic tissue, small foci of lymphocytes and polymorphs in the underlying fibrous tissue, and moderate hyperplasia of the lymphoid nodules of the glandular areas, but in one stomach there was rather more diffuse polymorph infiltration of the fibrous tissue and necrotic epithelium. In one other case some areas were obscured by massive haemorrhage arising deeply in the fibrous tissue at the level of the muscular coats.

The general impression was one of primary non-infectious ulceration, possibly with secondary bacterial infection in one instance.

Kidney: The kidneys showed a varying degree of

vacuolation and degeneration of the convoluted tubules. Eosinophilic spheres of plasma-like material were numerous in the lumina of the convoluted tubules and in the thin portion of the loops of Henle, and could occasionally be seen in Bowman's space. A few small foci of lymphocytes, were grouped principally around the smaller blood vessels. There was no evidence of the deeply eosinophilic granular casts described by Obel (1953) in hepatosis diaetetica, nor any lesion suggestive of a haemolytic crisis.

(c) *Chemical Examinations.*

Analysis of samples of stomach contents, livers and kidneys from dead pigs proved negative for evidence of poisoning by the more common toxic substances, including arsenic and lead, but because of the *post-mortem* picture, the possibility of copper poisoning was considered and estimation of the copper content of the liver from the first carcase received showed the very high value of 2,200 p.p.m. on a dry matter basis. Subsequently, copper estimations were carried out on liver samples from nine fatal cases, kidney samples from 5 fatal cases, the meal as fed to the pigs and on the purchased concentrate mixture. In addition, estimations were done on liver from eight pigs slaughtered at bacon weight and blood samples were also obtained from 6 of these pigs for estimation of copper. The results are summarised in Table 1.

TABLE I
COPPER CONTENT OF TISSUES FROM PIGS WHICH DIED OR WERE SLAUGHTERED

	Reference	Liver (p.p.m./ D.M.)	Kidney (p.p.m./ D.M.)	Blood (mg. per 100 ml.)
Pigs which died	1	2,200	950	—
	2	1,600	—	—
	5	1,400	—	—
	6	2,100	310	—
	7	1,400	280	—
	8	1,600	270	—
	9	1,100	230	—
	10	710	—	—
	11	1,600	—	—
Bacon pigs slaughtered	1	600	—	0·13
	2	620	—	0·19
	3	880	—	0·15
	4	1,300	—	0·14
	5	1,600	—	0·15
	6	1,250	—	0·15
Bacon pigs condemned	1	1,700	—	—
	2	2,100	—	—

Relatively few normal tissue-copper values for pigs are reported in the literature, but Cunningham (1946) gives a mean value of 41 p.p.m. on a dry-matter basis for pig livers obtained in Britain, and a mean of 18.9 p.p.m. for ones obtained in New Zealand where pigs are fed largely skim-milk and pasture, which would provide a lower copper intake than the concentrate diets usually used in Britain. Figures for "normal" fattening pigs obtained at the Central Veterinary Laboratory, Weybridge (personal communication) ranged from 19 to 170 p.p.m. with most values falling within 40 to 50 p.p.m./D.M., with kidney values of 20 to 25 p.p.m./D.M. and blood values of 0.12 to 0.19 mg. per 100 ml. Moustgaard and Olsen (1951) report a normal serum copper range for non-pregnant sows of 0.155 to 0.218 mg. with a mean of 0.18 mg. per 100 ml.

It is obvious that the liver and kidney concentrations given in Table I are all very much higher than those reported for "normal" pigs and confirm that excessive amounts of copper had been ingested by the pigs in this herd. Most of the values are similar to those which have been found in cases of chronic copper poisoning in sheep (Bull, 1949; Ogilvie, 1954; Clegg, 1956; Pearson, 1956) and they are comparable with those reported by Gordon and Luke (1957) in their case of copper poisoning in pigs. The blood-copper values for the pigs sent for slaughter are, however, not above the common normal range for pigs even though the liver-copper concentrations were abnormally high, but by analogy with chronic copper poisoning in sheep and cattle, elevation of blood-copper would not be expected until clinical symptoms were apparent shortly before death.

A sample of the meal as fed to the pigs showed a copper content of 130 p.p.m. while the purchased concentrate mixture was found to contain 1,500 to 2,800 p.p.m. copper. Because of the errors inherent in obtaining evenly mixed samples, these figures must be only an approximation, but nevertheless are a good indication that the meal as fed to the pigs contained about 10 times the amount of copper normally present in pig meal. The amounts found in the purchased concentrate are similar to those expected in a "copperised" mineral mixture and it was confirmed that copper had been added to the purchased concentrate with the object of controlling scours in pigs. As the pigs had no access to any other food, it must be assumed that the excessive storage of copper resulted from the feeding of this meal.

In view of the extensive haemorrhages found at *post-mortem* examination, it was thought advisable to exclude as far as practicable any possibility of poisoning by Warfarin, even though this had not been used or kept on the farm. Samples of stomach contents from dead pigs were therefore fed to laboratory animals (mice and guinea-pigs) and both the meal as fed to the pigs and the purchased concentrate were fed to rats for a period of 9 days. No ill-effects were seen and when killed the animals were perfectly normal. These results suggest that the samples were not contaminated by Warfarin.

Discussion

The clinical history of the case and the results of the *post-mortem* and pathological examinations carried out leave little doubt that the deaths of the pigs in this herd were caused by ingestion of some toxic agent included in the meal fed to the pigs on the farm. Chemical analyses showed no evidence of poisoning by the more common toxic substances but confirmed that excessive absorption and storage

of copper had occurred and the possibility of chronic copper poisoning must be considered.

As the result of extensive observations on chronic copper poisoning in sheep in Australia, Bull (1949) has reported that the criteria by which this condition can be recognised in sheep are haemoglobinuria, haemoglobinaemia, icterus, necrosis of the liver, kidney dysfunction leading to ureamia and a copper concentration in the liver usually higher than 1,000 p.p.m. on a dry matter, fat-free basis. It is possible that pigs affected with the same condition might react rather differently and that a somewhat different assessment would be required for accurate diagnosis but, except for the brief report from Northern Ireland by Gordon and Luke (1957), no reference to the occurrence of chronic copper poisoning in pigs has been traced. In the circumstances, it appears justifiable to compare the findings in the present case with those in Gordon and Luke's report and with such poisoning in other animal species.

In their case of copper poisoning in the pig, Gordon and Luke (1957) found generalised icterus, orange-yellow coloration of the liver, enlargement of the spleen and of the kidneys (the cortex of the latter being studded with large dark haemorrhagic areas) and blood-staining of the urine; the affected pigs lived for three days or longer. The jaundice and orange-yellow discoloration of the liver are apparently identical to the changes seen in the series of deaths now reported but enlargement of the spleen was not a regular feature in these cases and no evidence of haemoglobinuria or of such extensive kidney damage was observed. The duration of illness was apparently much shorter as the majority of pigs were reported as being "found dead." The condemnation of carcases at the bacon factory because of jaundice was another feature common to both reports but the ulceration of the oesophageal area of the stomach and the extensive haemorrhages, which were a frequent cause of death in the present case, were not present in the cases in Northern Ireland.

In the case of losses in pigs now reported evidence of icterus, necrosis of the liver, kidney damage and copper concentrations in excess of 1,000 p.p.m. in the liver of all but one fatal case were found. This suggests a similarity to chronic copper poisoning as reported in sheep, though no evidence of a haemolytic crisis or of haemoglobinuria was found. The liver damage in the pigs must have occurred over a considerable period. The fact that partial and total condemnation of carcases because of jaundice was necessary in apparently healthy pigs killed for bacon and that at least one pig is known to have shown yellow discoloration of the skin for a period of about 3 weeks prior to death, indicates that the jaundice also developed over a considerable period. This suggests the possibility that, in the presence of dangerous amounts of copper in the liver, the pigs may have been developing jaundice over a long period as the result of a gradual release of copper, instead of the sudden acute haemolytic crisis which occurs in most sheep from a sudden outpouring of copper from the liver. This possibility, and the fact that extensive haemorrhage was frequently the immediate cause of death, might account for the absence of lesions in the kidneys suggestive of a haemolytic crisis and of haemoglobinuria in the carcases of the pigs examined. The fact that no pig died which had not been fed on the copper-containing meal for a period of 8 weeks or longer, and that deaths stopped shortly after the change of feeding might lend further support to chronic poisoning as the cause of death.

A sample of meal as fed to the pigs was found to contain 130 p.p.m. copper on a dry matter basis and the pigs were fed at the rate of 3½ lb. dry meal per pig per day at the 100 lb. live-weight stage. At this level of feeding therefore, each pig would receive approximately 200 mg. copper each day and pigs of less or greater weight would receive a proportionately smaller or larger amount of copper daily depending on the amount of meal fed. However, as feeding was unrestricted up to 100 lb. live-weight and as a number of pigs were kept together in each pen in the fattening house, it is not possible to estimate accurately the food or copper intake of each animal. It is also possible that home-mixing of the food may not have been sufficiently thorough to ensure even distribution of the comparatively heavy concentrate mixture with the lighter cereals, and this may have resulted in the presence of "pockets" of meal containing higher amounts of copper than were found in the sample analysed. The supposition that uneven mixing may have occurred is suggested by the fact that the majority of deaths occurred some weeks after the commencement of home-mixing but the death in November 1955, and the condemnation of carcases in the batch of pigs slaughtered on December 17th, cannot be attributed to this cause. Moreover, the mixing on the farm had been carried out in accordance with instructions received with the machine at the time of installation.

Edgar (1942) reported that in sheep the addition of as little as 25 mg. of copper per day to a normal ration will raise the liver copper value to a dangerous level in 228 days and Naerland (1948) describes poisoning of sheep when fed 1 g. of copper sulphate for 100 days and also when sheep had free access for several weeks to salt licks containing 10 per cent. copper sulphate. Experimentally, Eden (1940) found that 1.5 g. of copper sulphate given daily induced chronic copper poisoning in sheep in 30 to 80 days, while Marston and Lee (1948) reported that 100 mg. copper per day, and to some extent 50 mg., was an unsafe dose.

There appears to be little information on the toxicity of copper for pigs but Carpenter, in the Annual Reports of the Hormel Institute of the University of Minnesota (1946-48), claimed that the addition of copper to the diet of pigs had a growth-promoting effect. In this country, Barber *et al.* (1955), in co-ordinated experiments at eight centres involving 182 pigs, reported that the addition of 250 p.p.m. copper in a mineral mixture to the diet of fattening pigs resulted in an improvement in growth rate and no untoward effects were observed. This level of copper is approximately double the amount found in the meal fed in the case now described. It is known,

however, that under some conditions sheep can have dangerously high liver copper concentrations without showing any clinical abnormality and the observations of Boughton and Hardy (1934) and of Bull (1949) indicate that stress factors such as fasting or physical exertion can precipitate the fatal haemolytic crisis in such sheep. Conditions which imposed any additional stress on pigs which had dangerously high liver copper concentrations could have been a contributing factor in the losses which occurred in the present case.

There are, therefore, close similarities in many respects to chronic copper poisoning as described in the literature. The very high liver and kidney copper values found in all pigs examined leaves no doubt that copper poisoning was, at the least, a potential hazard. Nevertheless, there are also certain features which are not fully consistent with a diagnosis of copper poisoning *per se* as the cause of the losses. The peculiar ulceration of the stomach and the extensive haemorrhages which occurred do not appear to have been observed in reported cases of chronic copper poisoning. The ulceration of the stomach is suggestive of some nutritional factor and the liver damage also is not inconsistent with such a conclusion. It may be that the lesions found in these pigs were due to the interaction of more than one factor and the presence of some unrecognised toxic agent in the diet cannot be excluded.

The ulceration which was present in the oesophageal area of the stomach in the majority of the carcases examined is very similar to that described by Obel (1953) and some 22 per cent. of naturally occurring cases of so-called toxic liver dystrophy (hepatosis diaetetica in swine, and fatal bleeding from perforation of arterial walls in the submucosa of the stomach was also observed in a proportion of these cases. However, in the course of experimental feeding trials, Obel found that the stomach ulceration was not characteristic of hepatosis dietetica as it occurred independently of the liver injury, and on diets which did not produce the liver changes. The ulceration was found to occur in pigs fed 6 per cent. cod liver oil in the experimental ration and it was thought to be produced by the unsaturated fatty acids in the cod liver oil in the absence of vitamin E. In the present case the diet did not contain cod liver oil, and should not have been deficient in methionine and cystine which protected against the development of liver damage in Obel's trials. The histological findings on livers from pigs in the present series of losses also suggested that the liver damage developed in a different way from that of hepatosis diaetetica and it is doubtful if this condition was involved.

With regard to the presence of other diseases, no evidence was found to suggest that bacterial infection might be the cause of death and the skin condition ("dry-feeding dermatitis") affecting pigs on the farm was much more prevalent in younger recently weaned pigs; lesions of this condition were found on one dead pig only. Many of the older fattening pigs were coughing, and pneumonic lesions were present in the lungs of some dead pigs but there was no evidence that pneumonia was involved as a cause of death.

It would probably be wrong at the present time to ascribe the deaths of these pigs to any one factor but consideration of the available evidence appears to indicate that, in addition to the excessive amounts of copper which were undoubtedly present in the carcases, some other unrecognisable toxic factor in the diet was also involved. The nature of the latter factor must, it is felt, await further investigation but the circumstances and findings of the case are thought worthy of record in view of the serious losses which occurred and in the hope that other workers may contribute from their experience.

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Effect of High Levels of Copper in Rations for Pigs

BY

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SUMMARY.—1. *Meals containing various levels of added copper sulphate were fed to pigs from weaning to bacon weight to study accumulation of copper in tissues, development of toxic symptoms and effects on growth rate.*

2. *No significant increase in live-weight gain was found on diets containing* 0.06, 0.10, 0.12 *and* 0.16 *per cent. added copper sulphate. Additions of* 0.2 *and* 0.4 *per cent. reduced growth significantly, caused jaundice and the death of* 3 *out of* 7 *pigs.*

3. *In spite of careful mixing, wide variations were found in the copper content of the meal at each level of supplementation.*

4. *Liver copper accumulation increased sharply on diets containing more than* 0.06 *per cent. added copper sulphate* (188 *p.p.m. Cu*) *but no toxic symptoms were produced on levels up to* 0.16 *per cent. copper sulphate.*

5. *At* 0.2 *and* 0.4 *per cent. levels toxic effects were produced. Three out of* 7 *pigs died, the remaining* 4 *were very unthrifty and were killed.*

6. *While the results suggest that high liver Cu levels* per se *do not necessarily produce toxic sequelae it appears that above a certain liver concentration, other factors may precipitate a syndrome attributable to Cu toxicity.*

INVESTIGATIONS by Buntain (1961) of the occurrence of deaths in a herd of pigs over several months in 1955-6, showed that liver copper values were exceptionally high (1,500 to 2,200 p.p.m. in dry matter (D.M.) and that a sample of the meal fed had a high copper content, 130 p.p.m. D.M. Some months prior to this investigation, reports (Barber, Braude & Mitchell, 1955; Bowler, Braude, Campbell, Craddock-Turnbull, Fieldsend, Griffiths, Lucas, Mitchell, Nickalls & Taylor, 1955) were published demonstrating that addition of 0.1 per cent. copper sulphate supplying 250 p.p.m. copper to a fattening pig diet increased growth rate significantly. No data on the copper content of liver or other tissues from pigs used in these experiments were reported but no harmful effects were associated with the addition of 0.1 per cent. copper sulphate to the ration.

At that time there was little information on copper toxicity in pigs or indeed, on the accumulation of copper in tissues from pigs fed rations to which different levels of copper had been added. As a complementary investigation to that carried out by Buntain (1961), a supply of the meal fed to the pigs in which the outbreak of suspected copper poisoning occurred, was obtained for a feeding trial. The purpose of this was to compare the effect on growth rate, accumulation of copper and possible development of toxic symptoms in pigs with that obtained from meals containing various levels of added copper sulphate. Although this work was done some years ago and on a small scale, it is thought that a description of the results would be of value. Cases of suspected copper poisoning in pigs are reported from time to time but there is little information on levels of dietary copper which cause excessive accumulation in pigs and the apparent variability of their tolerance to high tissue concentrations.

Experimental

Weaned Large White pigs of both sexes, 8 to 10 weeks old at the beginning of the experiment were used. They were housed in concrete pens in groups of 4 per pen except in one instance when a group of 3 pigs was used. Before experimental feeding started each pig was weighed, given 5 ml. of crystal violet vaccine and allocated at random to each treatment. The composition of the basal meal to which the various supplements of copper sulphate were added, is given in Table I. This was fed dry to appetite in troughs; there was one communal trough per group and a constant supply of water was available. The amount fed to each group was measured to allow an estimate of food intake per day. The pigs were reweighed at the end of the experimental period when they had attained bacon weight approximately 5 months after feeding began. Blood, liver and kidney samples were taken for estimation of copper. Samples of the meal as fed to the various groups were taken at intervals. Copper was estimated by a modification of the method of Eden and Green (1940).

TABLE I
PERCENTAGE COMPOSITION OF THE BASAL DIET

Barley meal	65
Middlings	25
White fish meal	10

Experiment I

The following diets were fed to 5 groups of pigs: (1) basal meal (control), (2) the meal used in the outbreak of suspected copper poisoning described by Buntain (1961), (3) basal meal plus 0.1 per cent. $CuSO_4.5H_2O$, (4) basal meal plus 0.2 per cent. $CuSO_4.5H_2O$, (5) basal meal plus 0.4 per cent. $CuSO_4.5H_2O$. These amounts of copper sulphate

when mixed with the meal should give concentrations of 250, 500 and 1,000 p.p.m. Cu respectively. There were 4 pigs in each group except (4) for which only 3 pigs were available.

Experiment II

Immediately following Experiment I, another 4 groups each containing 4 pigs were allocated to the same accommodation and fed the following diets: (6) basal meal (control), (7) basal meal plus 0.06 per cent. $CuSO_4.5H_2O$, (8) basal meal plus 0.12 per cent. $CuSO_4.5H_2O$, (9) basal meal plus 0.16 per cent. $CuSO_4.5H_2O$. The supplements were designed to provide concentrations of 150,300 and 400 p.p.m. Cu respectively. Blood samples were taken at intervals for haematological examination.

Results

Experiments I and II

The copper content of the diets, the estimated mean daily copper and food intakes and live-weight gains are given in Tables II and III.

normal progress. Group 5 (0.4 per cent. level) pigs ate little and grew very slowly. After one month on the diet all these pigs had died or were killed. The 3 pigs in group 4 (0.2 per cent. level) also ate little, grew slowly and jaundice occurred in 2; 1 of these died after about 3 months' feeding and the other 2 were so unthrifty that they were killed soon after. Group 2 (" Unknown " Cu) were affected by an undiagnosed febrile illness lasting about 2 weeks during the 4th month of feeding and involving the death of 1 pig; this illness caused a reduction of food intake during the 2-week period. A somewhat similar but less severe illness occurred in group 3 (0.1 per cent. level). These effects were reflected in reduced total food and copper intakes for groups 2, 4 and 5 (Table II). In spite of the reduction in food intake in group 2, there was no significant difference in live-weight gains in groups 1, 2 and 3; the poor growth of pigs in groups 4 and 5 was marked and the difference highly significant ($P = 0.001$).

No illness occurred in the pigs in Experiment II

TABLE II

EXPERIMENT 1. COPPER CONTENT OF DIETS, MEAN DAILY COPPER AND FOOD INTAKES AND MEAN LIVE-WEIGHT GAINS DURING THE FEEDING PERIODS FOR THE GROUPS

Group number	1	2	3	4	5
Per cent. $CuSO_4.5H_2O$ added	None	Not known	0·1	0·2	0·4
Number of samples analysed	7	6	10	7	3
Cu content p.p.m. in dry matter : Mean	7·4	239	279	518	1,100
Range	6·7–8·4	170–290	130–390	350–620	1,000–1,200
Feeding period (weeks)	21	21	21	14	4
Estimated daily Cu intake, mg. per pig	14·8	360	560	380	934
Estimated daily food intake, lb. per pig	5·2	3·9	5·2	1·9	2·2
Daily live-weight gain, lb. per pig	1·24	1·02	1·18	0·36	0·09

TABLE III

EXPERIMENT 2. COPPER CONTENT OF DIETS, MEAN DAILY COPPER AND FOOD INTAKES AND MEAN LIVE-WEIGHT GAINS DURING THE FEEDING PERIODS FOR THE GROUPS

Group number	6	7	8	9
Per cent. $CuSO_4.5H_2O$ added	None	0·06	0·12	0·16
Number of samples analysed	7	8	17	11
Cu content p.p.m. in dry matter : Mean	7·7	188	316	460
Range	6·1–9·6	150–220	220–390	330–600
Feeding period (weeks)	19	19	19	19
Estimated daily Cu intake, mg. per pig	14·1	345	579	843
Estimated daily food intake, lb. per pig	4·75	4·75	4·75	4·75
Daily live-weight gain, lb. per pig	1·15	1·22	1·26	1·24

Copper content of supplemented diets. Although copper sulphate was added to the basal meal in amounts to give concentrations of 150, 250, 300, 400, 500 and 1,000 p.p.m. in the diets of groups (7), (3), (8), (9), (4) and (5) respectively, Tables II and III show that a considerable variation in copper content was found in samples taken for analysis at intervals throughout the feeding periods, especially for groups (3) and (9). This was attributed to uneven mixing of the copper sulphate supplements with the meal.

Duration of high copper intakes and live-weight gains. It was not possible to maintain all the pigs on their allocated diets until bacon weight was reached since some groups of pigs failed to maintain and there was no significant difference in live-weight gains in groups 6, 7, 8 and 9 compared either with each other or with groups 1, 2 and 3, except that the gain in group 8 was just significantly greater than in group 2.

Post-mortem findings. The 2 pigs in group 5 which died after about 4 weeks on the 0.4 per cent. diet showed a marked orange discoloration of the liver. Both had a haemorrhagic gastro-enteritis and in the stomach of one there was a white necrotic area around the oesophageal opening. Some small haemorrhages were present in the kidneys of the other pig. The remaining 2 pigs in this group, which were killed about the same time, showed no abnormalities.

TABLE IV
EXPERIMENTS 1 AND 2. EFFECT OF DIETARY COPPER SUPPLEMENTS ON MEAN COPPER CONTENT OF LIVERS, KIDNEYS AND BLOOD OF PIGS

Group	Per cent. $CuSO_4 . 5H_2O$ added	Mean Cu content of diet, p.p.m./D.M.	Copper in tissues, p.p.m./D.M.			Blood copper, mg. per 100 ml.
				Liver	Kidney	
Experiment 1						
1	None	7·4	Mean	101	23·0	0·132
			Range	(23–170)	(20–24)	(0·12–0·14)
2	Unknown	239	Mean	862	38·4	0·142
			Range	(290–1,550)	(23–66)	(0·12–0·15)
3	0·1	279	Mean	1,725	37·8	0·140
			Range	(1,400–2,400)	(28–46)	(0·11–0·15)
4	0·2	518	Mean	4,167	793	0·265
			Range	(4,000–4,500)	(650–1,000)	(0·20–0·23)
5	0·4	1,100	Mean	2,725	1,228	0·860
			Range	(2,300–3,000)	(620–1,800)	(0·47–1·60)
Experiment 2						
6	None	7·7	Mean	93·5	29·3	0·128
			Range	(56–160)	(25–33)	(0·11–0·15)
7	0·06	188	Mean	168	34	0·113
			Range	(32–270)	(30–37)	(0·10–0·13)
8	0·12	316	Mean	1,525	55·3	0·128
			Range	(1,000–1,900)	(47–64)	(0·12–0·13)
9	0·16	460	Mean	2,575	107	0·135
			Range	(2,000–2,900)	(71–190)	(0·13–0·14

Orange discoloration of the liver was also marked in all 3 pigs in group 4 on the 0.2 per cent. diet. In the pig which died after 13 weeks there was a haemorrhagic gastro-enteritis. The other 2 pigs in this group were killed the following week; in the stomach of one there was ulceration of the oesophageal region; the other was normal.

It was notable that while the orange discoloration of the liver occurred in 2 of the pigs which were killed as well as in the 3 pigs which died, haemorrhagic gastro-enteritis was found only in the pigs which died.

Copper content of tissues. Values for copper content of livers, kidneys and blood are given in Table IV. There is no significant difference between liver copper values for the 2 control groups 1 and 6 and group 7. The values for all the other groups are very significantly higher ($P = 0.001$) than the values for these 3 groups. Values for groups 3 and 8 do not differ significantly from each other, nor do the liver copper values for groups 5 and 9. In spite of the similarity in liver copper concentrations of groups 5 and 9, the pigs in the former group did not survive longer than 4 weeks while none of those in the latter group showed any illness during 19 weeks. Highest liver copper concentrations were found in the 3 pigs in group 4; 1 died and the other 2 were killed because of extreme unthriftiness after about 3 months.

The mean kidney copper values for groups 8 and 9 are not significantly different from each other but are significantly greater than the values for groups 1, 2, 3, 6 and 7 among which there is no significant difference; the values for groups 4 and 5 are outstandingly high.

Blood copper values for all groups except 4 and 5 were within the range accepted as normal for pigs and showed no significant differences; those for groups 4 and 5 are obviously much higher than all the others ($P = 0.001$). No haematological abnormality was observed in any group.

Discussion

The results show that under the conditions of this experiment no significant increase in live-weight gains resulted from additions of 0.6, 0.10, "unknown" meal approximately 0.10, 0.12 and 0.16 per cent. copper sulphate to a basal meal for pigs fed from weaning to bacon weight; additions of 0.2 and 0.4 per cent. reduced growth rate very significantly ($P = 0.001$). Because of the small number of animals used in these 2 experiments, the failure to find a beneficial effect on growth is not unexpected. The necessity of having a large number of replicates in trials of this kind in which only a relatively small response to treatment is obtained has been pointed out (Bowler *et al.*, 1955). But the purpose of these 2 experiments was not to investigate the growth-promoting effect of various additions of copper sulphate to the diet of growing pigs; it was to gain some information on levels of copper in the diet which would produce abnormal accumulation of copper in the tissues, and on possible toxic effects resulting therefrom.

In spite of the small number of pigs in each group, it is clear that toxic effects were produced

by addition of 0.2 and 0.4 per cent. copper sulphate providing mean dietary copper concentrations of 518 and 1,100 p.p.m. respectively in the diet; 3 of the 7 pigs in these 2 groups died and the other 4 were so unthrifty they had to be killed. The concentration of copper in the livers of the pigs in group 4 on the 0.2 per cent. level of copper sulphate was much higher than that of pigs in group 5 on the 0.4 per cent. meal, yet toxic effects appeared more quickly and were more severe in the latter group. This suggests that it is not the concentration of copper *per se* in the liver, or in the kidney, or in both these tissues which caused the toxic effect. The pig in group 4 which died had liver and kidney copper concentrations of 4,000 and 1,000 p.p.m. respectively and 1 of the 2 in group 5 which died had much lower concentrations of 2,300 and 620 p.p.m. in the same tissues respectively. It would therefore appear that there is no common critical concentration of copper in these 2 tissues at which toxic effects might occur but there may well be individual critical levels which vary considerably for different animals. It is remarkable, too, that liver copper values for group 9 which maintained a good growth rate on a diet containing 0.16 per cent. copper sulphate did not differ significantly from group 5 in which growth rate was lowest and toxic symptoms were greatest.

Barber, Braude, Mitchell, Rook and Rowell (1957) found that the daily intake of food by 2 groups of 4 pigs fed meal containing 0.5 or 1.0 per cent. copper sulphate, rapidly fell to a very low level and the animals failed to gain weight over a period of 3 to 5 weeks. The meal was fed dry and *ad lib.*, unrestricted water being available in separate troughs. None of these 2 groups of pigs apparently suffered any permanent injury, as they resumed normal growth and food intake when the copper supplementation was reduced to the 0.1 per cent. level. The copper contents of liver and kidney tissues of these pigs at the end of the 50-day feeding period were not reported.

Comparison of the results obtained for group 5 on the 0.4 per cent. level of copper sulphate with those reported by Barber *et al.* (1957) suggests two possibilities. Either the level of copper supplementation which is high enough to be so unacceptable to pigs as to cause a sufficient reduction in food intake to enable them to escape toxic effects is a very critical one with a very narrow margin of safety, or there are factors other than the copper concentrations of the diet and the tissues which can cause toxic effects once liver and/or kidney copper accumulation has reached a certain critical level. The latter possibility would appear to be the most likely since it is well known that sheep can tolerate very high liver copper concentrations (1,000 to 3,500 p.p.m.) without showing any signs of toxicity until some form of stress is imposed on them after which a haemolytic crisis and death may rapidly follow (Bull, 1949; Marston, 1952). The observations recently reported by O'Hara, Newman and Jackson (1960) support this hypothesis. They found that a syndrome thought to be chronic copper poisoning occurred in pigs after about 16 weeks on a basal diet plus 0.1 per cent. copper sulphate. Four pigs died suddenly and one recovered after a period of illness; in all cases the clinical signs appeared during and immediately after driving the pigs from the yards to the weighing scales. Buntain (1961) suggested that conditions of stress superimposed on pigs whose tissues had accumulated excessive amounts of copper might have contributed to the deaths which occurred in the syndrome he described.

A comparison of the *post-mortem* findings with those reported by Buntain (1961), O'Hara *et al.* (1960) and Gordon and Luke (1957) shows that discoloration of the liver (yellow, orange or brown) was found in some pigs in all 4 investigations and jaundice in all but that reported by O'Hara *et al.* Gastro-enteric haemorrhage and ulceration of the oesophageal region of the stomach were present in our investigation and that of Buntain and absent in those of Gordon and Luke and O'Hara *et al.* Kidney haemorrhages described in the latter 2 reports were found in one of our pigs but were not observed by Buntain. A notable feature reported only by Buntain was the occurrence of subcutaneous haemorrhages in the legs of 3 pigs.

There is considerable variation in accumulation of copper in pigs fed copper-supplemented meals. Allen, Barber, Braude and Mitchell (1958) found a range of 317 to 2,560 p.p.m. copper in the livers of 12 pigs individually fed at the 0.1 per cent. level of copper sulphate supplementation. Lucas and Calder (1957) investigated the effect of diets supplemented with 0.012, 0.025, 0.05, 0.1 and 0.2 per cent. copper sulphate and individually fed wet to groups of 6 pigs from 8 weeks of age to slaughter at 200 lb. live-weight. At levels of 0.012 and 0.025 per cent., liver copper concentrations remained within the range Lewis (unpublished data) has found for normal pigs (16 to 48 p.p.m. dry matter; mean 29.62; S.E. $\pm$ 2.96); at the 0.05 per cent. level there was slight accumulation of copper, the mean value being 163 (range 37 to 540 p.p.m.). Above this level of supplementation, liver copper values rose sharply and at the 0.1 per cent. level mean accumulation was between 3 and 4 times greater; at the 0.2 per cent. level, mean accumulation was just over 5 times greater than the 0.1 per cent. level but the range was very wide (1,706 to 4,668 p.p.m., mean 3,085 p.p.m.). No signs of toxicity were reported but reductions in rate of gain and feed efficiency at the 0.2 per cent. level suggested marginal toxicity. Over the total experimental period, rate of gain and feed efficiency were highest on the 0.05 per cent. copper sulphate level which would give a concentration of about 125 p.p.m. copper in the meal.

Indications of marginal toxicity and liver copper levels of up to 3,000 p.p.m. were reported by Dammers and van der Grift (1959) in pigs fed wet meal containing 0.3 per cent. copper sulphate. Two of these livers had a greyish-brown appearance and showed some connective tissue proliferation. The pigs on this ration grew slowly. At levels of supplementation of 0.2 per cent. and below, no toxicity was apparent. At the 0.075 and 0.1 per cent. level

there was increased growth rate, appetite and dressed carcase percentage, whereas these increases were less marked at a level of 0.05 per cent.

The liver copper concentrations given in Table IV show a trend similar to that found by Lucas and Calder (1957). At the 0.06 per cent. level, values are of the same order as those at the 0.05 level used by Lucas and Calder, and *above* this level, a similar sharp increase in concentration occurred.

Although there is now much evidence that meals supplemented with copper sulphate at the 0.1 per cent. level have not only caused no untoward effects, but have increased rate of growth in fattening pigs, it appears that under some circumstances this level can cause a syndrome similar to chronic copper poisoning (Gordon & Luke, 1957; O'Hara *et al.*, 1960; Wallace, McCall, Bass & Combs, 1960; Buntain, 1961). Even though these occurrences are few, it should be recognised that excessive concentrations of copper in the tissues of pigs can represent a potential, although in most instances only a slight, hazard. Braude (1957) has rightly pointed out that the advantages obtained from the 0.1 per cent. level of supplementation should always be weighed against the hazards that might be involved. But if an equal growth-promoting effect is obtained from a diet supplemented at an 0.05 per cent. level as shown by Lucas and Calder (1957) without causing excessive storage of copper in liver and kidney tissues, there would appear to be little justification for the continued use of the 0.1 per cent. level. Indeed, a very large proportion of the tonnage of compounded pig meals sold in Britain contain copper sulphate at the 0.05 per cent. level. At present opinions appear to be divided on whether there is a real overall difference in growth-promoting effects between these 2 levels of supplementation.

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The Effect of Different Types of Pasture on Metabolic Disorders in Sheep

BY

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SUMMARY.—Controlled grazing experiments over a period of 3 years gave significantly lower mean serum magnesium values among sheep grazing actively growing herbage than in sheep grazing similar herbage with a run back on to a slower growing indigenous sward. There was a highly significant difference between the magnesium levels in individual animals and also between samples taken at different times. The percentage of animals with low serum magnesium values on the actively growing herbage was greater than on the mixed pasture in each year. The overall variation from year to year was not significant.

No significant difference was found between the mean values of serum calcium or of blood inorganic phosphorus.

There was a marked difference in the mean serum calcium levels from year to year, the lowest occurring in 1958.

There was still greater variation in mean blood inorganic phosphorus levels from year to year, the level being lowest in 1959.

One clinical case only, and 2 sudden deaths attributed to metabolic disorder, occurred, despite low individual values recorded. These occurred on rapidly growing herbage plots.

IN 1956 Stewart reported fairly widespread losses in hillflocks in Scotland due to hypomagnesaemic tetany. Since then the disorder has become increasingly common in both hill and lowland flocks. Incidence is greatest in ewes during the 4 weeks or so after lambing. One of the factors associated with the disorder appears to be the grazing of improved fertilised pastures or leys at the time of rapid growth when the fibre content is low and protein content high. Inglis, Weipers and Pearce (1960) reported a possible correlation between hypomagnesaemia and these two herbage constituents although only mild hypomagnesaemia and no clinical symptoms occurred in their ewes.

Among ewes under field conditions, metabolic upsets include changes in the blood levels of calcium, magnesium and inorganic phosphorus. It is impossible on clinical grounds alone to differentiate these conditions. An earlier survey of metabolic disorders by the writer (1955) disclosed within flock differences an eightfold increase in clinical disorder among sheep grazing short dense green pasture, compared with others on more mature herbage. It was therefore decided to investigate in greater detail the method of grazing young pasture with a run back to more mature herbage of wider nutritive ratio.

For experimental purposes a simple plan was adopted in which 2 groups of ewes with lambs at foot were introduced to adjoining plots, one providing actively growing herbage, the other with similar

herbage but including more mature yet palatable pasture. The object was to record any differences that arose in clinical behaviour, and in the blood levels of magnesium, calcium, acetone and inorganic phosphorous over the period of spring and early summer grazing.

Experimental Methods and Materials

An old pasture was divided into 2 equal sections, each 3½ acres. During 1956 the whole of section 2 and half of section 1 were directly reseeded, the remaining permanent pasture forming half of section 1 was retained. The seeds mixture sown at the rate of 22 lb. per acre consisted of S.23 perennial ryegrass 7 lb.; S.24 perennial ryegrass 5 lb.; commercial ryegrass 5 lb.; Italian ryegrass 3 lb.; S.100 white clover 2 lb.

Soil analysis showed an adequate lime status, very low potash and low phosphate contents. A compound granular fertiliser supplying a total of N 36 lb., K_2O 45 lb., P_2O_5 33 lb. per acre was therefore applied. This was considered a conventional dressing and in no way excessive. This top dressing was repeated annually on the whole of pasture 2 and on the reseeded half of pasture 1; the old sward forming part of this pasture was not treated. Pastures were close grazed prior to top dressing with fertiliser, which was applied approximately 3 weeks before the commencement of grazing. In all, the grazing period commenced about mid-April and lasted for a minimum period of 6 weeks. Spring drought delayed the establishment of the new sward in 1956, and grazing trials were commenced in 1957, being repeated during 1958 and 1959.

Welsh ewes previously mated to Radnor tups were lambed down during March each year, on adjoining fields of old pasture; the ewes were 5 and 6 years old at lambing time. Lambs were weighed and identified with their dams at birth. It was possible later, by re-weighing the lambs before the trials commenced, to form some estimate of the milking capacity of the ewes. Ewes with twin lambs were accommodated separately until required for the grazing trials. Two groups of sheep from the main flock were matched as evenly as possible for age, date of lambing, condition and weight, and milking capacity; ewes with twin lambs were equally apportioned to either group. The total number of ewes employed for 3 successive years was 42, 38 and 34 respectively. The average age of lambs at the commencement of each trial was 26, 23 and 25 days.

Pasture samples were restricted to one sample from each pasture at the commencement of every grazing trial, for sampling can present a considerable source of error due to two reasons: the animal's predilection for meristematic tissue, and the fact that the ground covered with a particular grass species may be disproportionate to the amount consumed. Blood samples were taken immediately before, and at intervals throughout the grazing period. Following the random introduction of ewes to the chosen sections, the animals were left undisturbed. The long interval between first and second blood samples in 1957 was adopted to eliminate the possibility of undue handling precipitating clinical cases of tetany. This was thought to be unnecessary and the interval was shortened in subsequent trials. When necessary, additional sheep were added to pasture 2 (reseeded) to regulate growth of herbage as compared with pasture 1 (mixed). These additional sheep were not included in the results.

Results

Table I shows the mean serum magnesium, calcium, and inorganic phosphorus values for all blood samples taken over the 3-year period. All the mean magnesium levels for animals on the reseeded pasture

TABLE I
MEAN VALUES OF SERUM MAGNESIUM, CALCIUM AND INORGANIC PHOSPHORUS (AND RANGE OF INDIVIDUAL VALUES) FOR EWES RUNNING ON :—
(1) Mixed pasture, half consisting of a reseeded portion, half of old pasture. (2) Reseeded pasture only.

Date	Means value (mg. per 100 ml.)					
	Pasture (1)	Pasture (2)	Pasture (1)	Pasture (2)	Pasture (1)	Pasture (2)
	Mg		Ca		I.P.	
11.4.57	1·96	1·91	10·49	10·36	3·81	3·35
23/24.4.57	2·32	2·00	10·06	9·70	3·42	3·16
20/21.5.57	2·19	2·14	10·36	10·36	4·41	4·04
Mean for 1957 ...	2·16	2·02	10·30	10·08	3·88	3·52
Range	1·3–3·0	0·7–2·9	8·2–11·8	8·6–11·4	1·1–6·1	1·3–5·7
15.4.58	2·19	2·13	8·88	9·54	4·14	4·03
23.4.58	1·88	1·70	9·06	8·74	3·87	3·88
8.4.58	2·11	1·96	9·51	9·42	4·99	5·03
6.6.58	2·21	2·08	9·95	9·64	4·56	4·45
Mean for 1958 ...	2·10	1·97	9·35	9·33	4·39	4·35
Range	0·7–3·6	0·6–3·0	6·7–10·8	6·8–11·2	2·0–6·8	1·8–6·7
15.4.59	2·26	2·05	9·76	9·92	2·11	3·22
20.4.59	2·17	1·82	9·50	8·98	2·84	2·75
30.4.59	2·17	2·07	10·51	9·97	3·49	3·40
27.5.59	2·34	2·34	10·15	9·80	4·21	4·38
Mean for 1959 ...	2·24	2·07	9·98	9·67	3·18	3·44
Range	0·5–2·8	0·6–2·9	7·8–11·8	7·1–10·9	0·6–5·6	1·3–5·9

2 are lower than the corresponding values for animals on the mixed pasture 1, consisting of a reseeded portion and an old portion; this difference is significant ($P = 0.05$). There is a highly significant difference ($P = 0.01$) between the magnesium levels in individual animals and also between samples taken at different times, but the overall variation from year to year is not significant.

Mean calcium values for animals on the reseeded pasture 2 are almost all lower than those on the mixed pasture 1, but the difference between the 2 groups is not significant. There is, however, a marked difference in calcium levels from one year to another, both groups being lower in 1958 than in the other two years, and this difference is highly significant ($P = 0.01$).

TABLE II
PERCENTAGE OF ANIMALS (FOR ALL SAMPLINGS) HAVING ABNORMALLY LOW LEVELS OF SERUM MAGNESIUM, CALCIUM AND INORGANIC PHOSPHORUS

	Year	Pasture (1) mixed section	Pasture (2) reseeded section
Magnesium (less than 1·8 mg. p.c.)	1957	19·4	22·2
	1958	23·3	32·4
	1959	10·3	22·1
Calcium (less than 8·5 mg. p.c.)	1957	1·6	0·0
	1958	11·1	14·1
	1959	4·4	4·4
Inorganic Phosphorus (less than 3·5 mg. p.c.)	1957	26·8	52·8
	1958	14·3	12·9
	1959	64·2	57·4

Inorganic phosphorus shows greater variation than either magnesium or calcium. There is no significant difference between animals on the two types of pasture, but again there is a highly significant difference from year to year, the level being lowest in 1959.

The percentage of animals in which serum magnesium, calcium and inorganic phosphorus fall to an abnormally low level is shown in Table II. The percentage of animals with low magnesium on the reseeded pasture is greater than that on the mixed pasture in each year; there is very little difference in the percentage of animals with low calcum on the 2 pastures in any one year, or for those with low inorganic phosphorus (except in 1957). Differences between the years for both calcium and inorganic phosphorus on both pastures are greater than those for magnesium, the greatest percentage of animals with low calcium values occurred in 1958, and with low inorganic phosphorus values in 1959.

No clinical case of tetany or death was recorded on pasture 1 (mixed) throughout the periods of grazing. On pasture 2 (reseeded) one clinical case of tetany occurred in 1957, and 2 sudden deaths were recorded in 1958. The clinical case occurred 9 days after the commencement of grazing, the ewe having lambed 26 days earlier. The sudden deaths 7 and 11 days following the commencement of grazing were in ewes with lambs 29 and 37 days old respectively. The deaths were attributed to metabolic disorder. Table III records the average liveweight gains for ewes and lambs on both sections.

The data indicate that reasonably even conditions

TABLE III
AVERAGE LIVE WEIGHT GAINS FOR EWES AND LAMBS

	Pasture (1) (Mixed)				Pasture (2) (Reseeded)			
	1957	1958	1959	Average for 3 years	1957	1958	1959	Average for 3 years
Initial weight ewes (lb.) ...	82	70	72·4	74·8	84	70	73	75·7
Liveweight gain ewes (lb.) ...	8	5	8·2	7·1	6	6	6·5	6·2
Initial weight lambs (lb.) ...	26	17·6	20	21·2	24	17·5	20·5	20·7
Liveweight gain lambs (lb.) ...	24	29·4	22·3	25·2	26	29·5	23	26·2

TABLE IV
ANALYSIS OF PASTURE SAMPLES

	Crude protein	Crude fibre	Digest crude protein	Nutritive ratio*	K % DM	Mg % DM	Ca % DM	P % DM
Pasture (1) (Reseeded)								
1957 ...	24·4	18·6	20·3	1 : 2·8	—	0·18	0·97	0·94
1958 ...	25·3	14·9	21·0	1 : 2·7	1·97	0·30	1·08	1·05
1959 ...	27·7	15·6	23·5	1 : 2·3	3·75	0·30	0·92	1·20
Pasture (2) (Mixed) *Reseeded Area*								
1957 ...	28·0	19·4	24·1	1 : 2·2		0·16	0·98	0·92
1958 ...	28·6	15·8	24·9	1 : 2·1	2·02	0·34	1·17	1·33
1959 ...	24·0	18·1	19·9	1 : 2·9	3·35	0·23	1·12	1·09
Old Grazing Sward								
1957 ...	22·3	17·3	18·5	1 : 3·2	—	0·21	2·2	0·64
1958 ...	18·8	18·0	14·7	1 : 3·5	1·51	0·32	1·24	0·71
1959 ...	19·3	17·5	15·4	1 : 4·0	2·4	0·23	1·37	1·55

* Nutritive ratio : $\frac{\%\text{ dig. crude protein}}{\%\text{ dig. carbohydrate} + (\text{dig. oil} \times 2{\cdot}3) + \%\text{ dig. fibre}}$

TABLE V
ESTIMATED BOTANICAL COMPOSITION OF PLOTS AT COMMENCEMENT OF GRAZING

	1957			1959		
	Pasture (1) Reseeded %	Pasture (2) Reseeded %	(Mixed) Old Sward %	Pasture (1) Reseeded %	Pasture (2) Reseeded %	(Mixed) Old Sward %
Italian Ryegrass	10	10	Trace	—	—	—
Perennial Ryegrass ...	55	65	10	25	40	10
Timothy	—	—	15	5	5	5
Other grasses	20	15	55	55	30	35
White Clover	Trace	Trace	10	5	15	20
Weeds	10	5	10	10	10	30
Bare ground	5	5	Trace	—	—	—

Other grasses : Yorkshire fog ; Bent ; Rough stalked meadow grass ; Animal meadow grass ; Crested dogstail.
Weeds : Creeping buttercup ; Sedge ; Daisy ; Silverweed ; Dock ; Creeping thistle ; Common rush ; Glyceria.

of grazing were maintained between plots. Table IV records the analytical results on pasture samples.

It may be noted that the old grazing sward had a wider nutritive ratio than both reseeded areas.

There were no marked differences in magnesium values between sections in any one year, but there was a marked difference in values between years. Such differences in pasture magnesium was not related to mean blood magnesium values since hypomagnesaemia was not more marked in 1957 than in the other 2 years. Table V gives an eye estimation of the botanical composition of the different swards at the commencement of grazing 1957 and 1959. There is evidence of reversion indicating but not proving that the manurial effects might have been greater than floristic differences between the plots. This was not carried out in 1958.

The clover contribution was small, as is more generally the case early in the year, particularly following hard autumn and winter grazing.

Other grasses, volunteer species, showed an increase in both reseeded areas in 1959.

Discussion

The better results were obtained on the mixed section and are reflected by the higher blood values, and absence of clinical cases and deaths. A significant difference in mean serum magnesium values was found between sections, under these experimental conditions. On the other hand, mean calcium and inorganic phosphorus values showed a significant difference, not between sections but between the years. It appears that factors other than those of sward difference are concerned. These may be related to climatic effects or to inherent physiological differences in the ewes since some fresh animals were introduced each year, although from year to year every practical attempt was made to maintain uniform conditions. Climatic conditions over winter varied considerably from year to year, although weather conditions during each period of grazing were reasonably even.

A wide range of individual variation in levels of magnesium, calcium and inorganic phosphorus values was found and the significance of experimental results obtained with very small numbers of sheep may be questionable. Despite the low individual values recorded, only one clinical case of tetanty and 2 sudden deaths were encountered throughout; all of these occurring on the reseeded section. It is probable that the onset of metabolic disorders is influenced by more than one factor. It is of interest to note that the one clinical case of metabolic disorder (Ca 2.8; mg 1.6; i.p. 2.6; mg 100 ml.) showed marked hypocalcaemia. Under field conditions concurrent hypocalcaemia with hypomagnesaemia occurs frequently. It is worth recording the subsequent behaviour of this animal following treatment and recovery. She was withdrawn from the experiment and placed in an adjoining plot where for some days she continued to graze indigenous slower growing herbage, avoiding young active pasture. This preference for indigenous herbage not recently top dressed with fertiliser has been observed on many occasions in similar recovering cases. Perhaps this behaviour indicates a digestive disorder associated with metabolic disorder.

Differences between plots are both floristic and manurial, in which the rapidly growing herbage provides a diet with a more narrow nutritive ratio than that of the slower growing old pasture. For an old sward to be beneficial it must be palatable; herbage can be so mature and fibrous as to prove unacceptable to the animal.

Earlier experimental work with metabolic disorders in relation to hill reclamation (1953) indicated that one possible function of more mature herbage was to alleviate the sudden change in diet imposed upon the sheep when suddenly transferred from poorer to greatly improved swards during the early spring.

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References

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B.V.A. Congress Programme

SUNDAY, September 3rd, 1961

Official or Scientific Programme

2.00 p.m. B.V.A. Office open (Wadham College).
4.30 p.m. Council Meeting.

Social Programme

6.00 p.m. Service at the Cathedral (Christ Church College).
8.00 p.m. Royal Counties Division Reception (till 10.00).

MONDAY, September 4th, 1961

Official or Scientific Programme

9.30 a.m. B.V.A. Congress Office open (Rhodes House).
10.00 a.m. Official Opening of Congress and Exhibitions.
11.15 a.m. Annual General Meeting.
12.30 p.m. Congress Group Photograph.
2.30 p.m. to 4.30 p.m. Symposium on Staphylococcal Mastitis.
The Immunity of Staphylococcal Mastitis (J. B. Derbyshire).
The Epidemiology of Staphylococcal Mastitis (I. Davidson).
Treatment (C. D. Wilson).
Chairman : I. H. Pattison.
Opener : S. J. Edwards.
2.30 p.m. to 4.30 p.m. Protection Against Canine Virus Diseases in Great Britain.
Speaker : W. Mansi.
Chairman : A. C. Dixon.
Opener : R. Hanscombe.
5.00 p.m. Films.

Social Programme

7.30 p.m. Civic Reception and B.V.A. Dance (till 1.00 a.m.).

Tours

Note : College tours available throughout the day.
1.45 to 6.00 p.m. Excursion to Huntley and Palmers, Reading (including Tea).

TUESDAY, September 5th, 1961

Official or Scientific Programme

9.00 a.m. to 11.00 a.m. Symposium on Recent Advances in the Treatment and Control of Intestinal Parasites.
Treatment of Domestic Animals.
Recent Advances in Anthelmintics (T. E. Gibson)
Mechanisms in Helminth Infections (E. J. L. Soulsby).
Chairman : N. S. Barron.
Opener : J. A. Pasfield.
9.00 a.m. to 10.00 a.m. Some Aspects of Dog Nutrition (R. Gordon Booth)
Chairman : G. N. Henderson.
Opener : A. N. Worden.
10.15 a.m. to 11.15 a.m. Further Experiences with Cage Birds (L. O. Arnall).
Chairman : R. H. Axworthy.
Opener : O. Graham-Jones.
11.45 a.m. to 12.45 p.m. Plenary Session. International Collaboration in Veterinary Matters (A. W. Stableforth) *Chairman :* S. L. Hignett.
2.30 p.m. Meeting of Association of Veterinary Food Hygienists.
2.30 p.m. to 3.30 p.m. The Biological Basis for Precautions in Veterinary Radiography (R. H. Mole)
Chairman : J. Deans Rankin.
Opener : W. D. Tavernor.
3.45 p.m. to 4.45 p.m. Diseases Associated with Broiler Rabbits (D. C. Ostler).
Chairman : H. A. Crawshaw.
Opener : S. T. Harriss.
5.00 p.m. Films.

Social Programme

7.15 for 7.45 p.m. Annual Banquet.
(Evening) Theatre Party, Oxford.

Tours

Note : College tours available throughout the day.
9.15 a.m. Excursion to M.G. Car Company Ltd. (including lunch).
11.30 to 6.00 p.m. Excursion to B.O.C.M., Stoke Mandeville.
2.30 to 6.00 p.m. Excursion to Blenheim Palace.

WEDNESDAY, September 6th, 1961

Official or Scientific Programme

9.00 a.m. to 10.00 a.m. Safe Use of Pesticides in the United Kingdom (E. J. Miller).
Chairman : J. W. R. Pearce.
Opener : J. L. McGirr.
9.00 a.m. to 10.00 a.m. Broiler Calves (J. Wilson)
Chairman : S. L. Hignett.
Opener : J. Makinson.
10.15 a.m. to 11.15 a.m. The Use and Misuse of Mineral Supplements (R. Allcroft).
Chairman : H. F. C. Hebeler.
Opener : J. A. A. Watt.
10.15 a.m. to 11.15 a.m. Variations in Susceptibility to Infection with *Mycobacterium johnei* (R. L. Chandler).
Chairman : T. C. Denholm.
Opener : E. J. H. Ford.
11.45 a.m. to 12.45 p.m. Plenary Session : Veterinary Surgery and Orthopaedics (J. Trueta). *Chairman :* S. Jennings.
2.00 to 6.45 p.m. Visit to Surgeries at Wallingford, Reading and Newbury (including Tea).
2.00 p.m. Visit to Grassland Research Centre, Hurley.
2.30 p.m. to 4.30 p.m. Pathogen-free Pigs for Research and the Practical Control of Pig Diseases (A. O. Betts).
Chairman : J. A. de Garis.
Opener : M. R. McCrea.
5.00 p.m. Films.

Social Programme

8.00 p.m. Ladies' Guild Dance (till 1.0 a.m.).
(Evening) Theatre Party, Oxford.

Tours

Note : College tours available throughout the day.
2.00 to 6.15 p.m. Excursion to Witney Blanket Factory and tour of the Cotswolds (including Tea).

THURSDAY, September 7th, 1961

Official or Scientific Programme

9.00 a.m. to 11.00 a.m. Symposium on the Provision of Laboratory Diagnostic Services.
Introduction (D. L. Hughes).
Structure and Availability (R. M. Loosmore).
Training of Workers (W. L. Weipers).
The Veterinary Surgeon in Practice and His Need of Laboratory Services (J. B. White)
Chairman : A. M. Graham.
9.00 a.m. to 11.00 a.m. Symposium on Equine Practice.
Clinical Observations on Chronic Lameness in the Horse's Foot (J. E. Gray).
Clinical Aspects of Equine Brucellosis (J. S. M. Cosgrove).
A Review of the Therapeutic Use of Corticosteroids in Equine Lameness (J. H. Wilkins).
Chairman : H. Bell.
Opener : A. C. Fraser.
11.45 a.m. to 12.45 p.m. Plenary Session : Recent Advances in the Control of Viral Epizootics by Vaccination Techniques (H. Frenkel.) *Chairman :* A. G. Beynon.
1.45 p.m. Visit to M.R.C. Unit, Harwell (returning 6.00 p.m.)
2.30 p.m. to 4.30 p.m. Symposium on Salmonellosis in Man and Animal.
Salmonellosis in Calves (E. A. Gibson).
Human Salmonellosis in Relation to Infection in Animals (N. S. Galbraith).
Chairman : R. Lovell.
Opener : A. Buxton.
5.00 p.m. Films.
6.00 p.m. International Radio Discussion on Pig Diseases.

Chairman :
Dr. N. O. Christensen—Veterinaer-og-Landbohojskole, Ambulatorisk Klinik, Bulowsvej 13, Copenhagen.

Members :
Professor N. O. Rasbech, Professor H. C. Bendixen } Veterinary College, Copenhagen.
Miss B. Knox-Seith—State Serum Laboratory, Copenhagen.

Social Programme

2.00 p.m. Golf Tournament, Frilford Heath.
(Evening) Theatre Party at Royal Shakespeare Theatre, Stratford-on-Avon (" Romeo and Juliet") and Alumnus Dinners.

Tours

Note : College tours available throughout the day.

FRIDAY, September 8th, 1961

Official or Scientific Programme

9.00 a.m. to 11.00 a.m. Symposium on the Role of Wild Life in the Dissemination of Disease.
General Introduction and Veterinary Problems (A. McDiarmid).
The Effect of Disease on Wild Mammals and Birds (H. V. Thompson).
The Role of Wild Life in the Epidemiology of Certain Human Diseases (R. H. A. Swain)
Chairman : W. S. Gordon.
Opener : A. R. Jennings.

9.00 a.m. to 11.00 a.m. Symposium on Some Modern Feeding Methods.
The Early Weaning of Lambs (C. Spedding).
Self-feed Silage (R. R. Turner).
The Zero-Grazing of Dairy Cows (K. B. Runcie).
Chairman : R. M. Loosmore.
Opener : S. L. Hignett.

11.45 a.m. to 12.45 p.m. Symposium on Tissue Grafting.
The Relevance to Veterinary Surgery of Modern Work on Tissue Grafting and Immunity (J. M. Payne).
Skin Grafting in the Treatment of Granulating Wounds of the Limbs of the Horse (P. A. Neal).
Chairman : L. G. Anderson.
Opener : P. M. Biggs.

2.00 p.m. Visit to A.R.C. Field Station, Compton (returning 6.30 p.m.).

2.30 p.m. to 4.00 p.m. Symposium on Poultry Diseases.
Clinical Diagnosis of Poultry Diseases (W. P. Blount).
The Control of Poultry Diseases (J. D. Blaxland).
Chairman : W. E. Beattie.
Opener : P. J. Dalton.

5.00 p.m. Films.

Social Programme

8.30 p.m. President's Reception (till 1.00 a.m.).

Tours

Note : College tours available throughout the day.
9.30 a.m. Excursion to Whipsnade (returning 6.00 p.m.).
2.00 p.m. River trip to Abingdon, returning by coach at 6.00 p.m.

SATURDAY. September 9th, 1961

Official or Scientific Programme

10.00 a.m. Adjourned A.G.M. and Close of Congress, followed by First Meeting of Council, 1961–2.

CONGRESS NOTES

Golf Tournament

Golf tournaments for the Simpson Trophy for men and the Dunkin Trophy for ladies will be played at Frilford Heath Golf Club (7 miles from Oxford on the Wantage Road) at 2.30 p.m. on Thursday, September 7th. The green fee will be 10s. per head and tea will cost 2s. 6d. Applications for entry, together with a remittance for 12s. 6d., should be made to: Mr. I. Meikle, 14, Queen's Road, Reading, Berkshire.

Theatre Parties, Oxford, September 5th and 6th

A block booking will be arranged at the New Theatre and/or Oxford Playhouse for each of the above evenings. Tickets will be available on application to the Congress Office at Oxford.

Theatre Party, Stratford-on-Avon, September 7th

The whole allocation of seats for the Stratford Memorial Theatre have been taken up and no further applications can be accepted.

Photography Exhibition

A meeting and exhibition of photography and cinematography will take place on Tuesday, September 5th.

Parking of Cars in Oxford

Parking of cars is difficult in Oxford but the City of Oxford police have undertaken to assist. A list of parking places will be given in the Congress programme which will include both day and night parking facilities and a windscreen sticker has been authorised. Space has been reserved for up to fifty cars at Messrs. Morris Garages Ltd., St. Aldates, Oxford, at a cost of 30s. for the week. Those wishing to avail themselves of this facility should make early application direct to the garage.

Excursion to Morris Motors

Owing to circumstances beyond the control of the organisers it has been found necessary to cancel the arrangements for the excursion to Morris Motors and, instead, to visit the M.G. car factory at Abingdon. Lunch arrangements and time of departure remain unchanged.

The names of those who applied to visit Morris Motors have been placed on the list to visit M.G. car factory and, unless hearing to the contrary by July 31st, it will be assumed that this new arrangement is acceptable.

Visit to M.R.C. Unit, Harwell

All tickets have now been sold for the visit to Harwell.

IMPORTATION OF CHAROLLAIS CATTLE

Seven farming experts flew to France on Monday to look for 30 Charollais bulls to improve Britain's beef. Mr. William Dodgson, chief livestock officer of the Ministry of Agriculture, leads the mission which includes veterinary experts and representatives of the Milk Board and private cattle breeding centres.

They will visit about eight farms near Nevers and Moulins to make a preliminary selection of about 50 six-month-old calves, expected to cost about £700 each.

The French farmers have agreed not to vaccinate the calves against foot-and-mouth disease, and to retain the animals for a final selection in October of the best 30. These will be held in quarantine on both sides of the Channel and distributed to cattle breeding centres during the winter.

They will be available, by artificial insemination, next spring to farmers who wish to try cross-breeding them with dairy cattle.

News and Comment

THE REGISTER OF VETERINARY SURGEONS

The undermentioned gentleman, having passed the final examination for the Diploma of the Royal College of Veterinary Surgeons, was admitted to Membership on July 7th, 1961:—

Oppong, Emmanuel Nana Waddie, P.O. Box 1110, Kumasi, Ghana.

The following were admitted to Membership of the Royal College of Veterinary Surgeons on July 8th, 1961, in virtue of the registrable degree of B.V.M.S., conferred on them by the University of Glasgow:—

Atang, Protus George, Veterinary Department, South Cameroons, West Africa; Crawford, Iain, 17, Faulds, Baillieston, Glasgow; Dabell, John Stewart, 23, St. Ives Avenue, Blackpool, Lancs.; Deans, Robert William, Cairnton, Fordoun by Laurencekirk, Kincardineshire; Ferguson, Donald Angus McPhie, 16, Blarbuie Road, Lochgilphead, Argyll; Galbraith, Brian David, 7, Croftburn Drive, Glasgow, S.4; Heeley, Rachel Mary (Miss), 85, Woodend Drive, Glasgow, W.3; McLelland, John, University Veterinary Hospital, Bearsden Road, Bearsden, Glasgow; Miller, David John Stanley, Chapel Hill, Dukinfield, Cheshire; Miller, Thomas Alexander, c/o McKie, 251, Kenmure Street, Glasgow, S.1; Mollison, Athole, 1, Seafield Road, Arbroath, Angus; Nyarko, Daniel, Pepiase, *via* Mampong Ashanti, Ghana, West Africa; Peek, Ian Southren, 27, Merryburn Avenue, Giffnock, Glasgow; Richards, Robert Stuart, Abingdon, Dunblane, Perthshire; Saunders, Robert Woolhouse, 17, Gold Street, Riseley, Bedford; Selman, Ian Edward, Old Stones Cottage, Ightham, nr. Sevenoaks, Kent; Todd, James Norman, 11, Lindsay Drive, Glasgow, W.2; Shaw, Duncan, 140, Welbeck Crescent, Troon, Ayrshire; Smeaton, Peter Dewar, Chelsfield, 9, Gregory Springs Road, Mirfield, Yorkshire; Wheeldon, Anne (Miss), 62, Speirs Road, Bearsden, Glasgow; Wishart, David Fergusson, Steephill, Drummond Terrace, Crieff, Perthshire; Wyburn, Robert Sharp, 12, Woodcraft Avenue, Glasgow, W.1; Young, Richard Gavin Ellis, Thackray House, Hatfield, Doncaster, Yorkshire.

PERSONAL

Dr. A. Ginsberg has been invited to give a series of lectures on meat hygiene at the University of Giessen from July 19th to 28th.

Dr. J. R. M. Innes will be visiting England from August 18th to November 1st and will be lecturing in Cambridge, Edinburgh, Glasgow and London. He will be participating in the International Congress of Neuropathology in Munich, and also in a following one of Neurology in Rome. Members of the profession who would like to contact Dr. Innes during this period are asked to address their letters c/o B.V.A., 7, Mansfield Street, Portland Place, London, W.1.

Mr. T. Stewart, M.R.C.V.S., D.V.S.M., General Manager of the Veterinary and Farm Sales Division of Bayer Products Ltd., Surbiton, has been appointed Managing Director of Phillips, Scott & Turner (Ireland) Ltd., and General Manager of the Bayer Products Division of Winthrop Group in Ireland. Both of these Companies are members of the Winthrop Group Ltd., and Mr. Stewart will be responsible for all the activities of that Group in Ireland.

Dr. W. Ross Cockrill, F.R.C.V.S., Chief of the Section of Veterinary Medicine in the Food and Agriculture Organisation of the United Nations, Rome, has been appointed Assistant Director, Animal Production and Health Division.

Engagement

HOPKINS—WHITELEGG.—The engagement is announced between David Lea Hopkins, B.V.SC., M.R.C.V.S., elder son of Mr. and Mrs. H. G. Hopkins of Congleton, Cheshire, and Jean Whitelegg, younger daughter of Mr. and Mrs. A. Whitelegg of Brereton, Cheshire.

Marriage

ROBINS—FORGAN.—On July 21st, 1961, at St. John's Presbyterian Church, Orpington, David Hilton Robins, B.VET.MED., M.R.C.V.S., to Janet Elizabeth Forgan.

Birth

BALL.—On June 11th, 1961, to Jenny, wife of David Ball, B.VET.MED., M.R.C.V.S., of 223, Dorchester Road, Weymouth, a daughter, Deborah Caroline.

COMING EVENTS

July

24th (Mon.) CENTRAL VETERINARY SOCIETY. Visit to the Post-graduate Medical School, Hammersmith, 2.30 p.m.

September

2nd (Sat.). STREATLEY '58 CLUB REUNION. Annual Dinner at "Ye Miller of Mansfield."

3rd to 9th (Sun. to Sat.). 79th Annual Congress and Exhibitions of the British Veterinary Association at Oxford

7th (Thurs.). ROYAL VETERINARY COLLEGE ASSOCIATION. Annual Dinner during the B.V.A. Congress at St. Edmund's Hall, Oxford.

GLASGOW VETERINARY SCHOOL ALUMNUS ASSOCIATION. Dinner at the Mitre Hotel, Oxford, 7 p.m.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the date on which disease has been confirmed, followed by the postal address and local authority.

Anthrax

July 10th, 1961. J. C. G. Barnes, Owlshay Farm, Buckland St. Mary, Chard, Somerset (Parish: Buckland St. Mary). (Cattle.) SOMERSET.

July 13th, 1961. W. Parris, Aller Farm, Up Ottery, Honiton, Devon (Parish: Up Ottery). (Cattle.) DEVON.

July 13th, 1961. V. S. Broom, Catshayes Farm, Gittisham, Honiton, Devon (Parish: Gittisham). (Cattle.) DEVON.

July 17th, 1961. J. H. & E. R. Swindell, Stoney Close Farm, Bakewell, Derbys. (Cattle.) DERBYSHIRE.

Fowl Pest

July 11th, 1961. Mrs. E. H. Barnes, The Homestead, The Avenue, Eye, Suffolk. EAST SUFFOLK.

July 11th, 1961. Mrs. T. Martin, 14, Council Houses, Wickham Skeith, Eye, Suffolk. EAST SUFFOLK.
July 11th, 1961. F. E. Webb, Hall Farm, Kelsale, Saxmundham, Suffolk. EAST SUFFOLK.
July 12th, 1961. P. Cross, Walmer Hall, Little Hoole, Preston, Lancs. LANCASHIRE.
July 12th, 1961. S. W. Saint, Birch Field, Wayside Road, St. Leonards, Ringwood, Hants. (HAMPSHIRE.
July 13th, 1961. C. W. Sheppard, Park View, Barham, Ipswich, Suffolk. EAST SUFFOLK.
July 13th, 1961. A. R. Seeley, Pauto, Victoria Hill, Eye, Suffolk. Disease at: The Gardens, Magdalen Street, Eye, Suffolk. EAST SUFFOLK.
July 15th, 1961. S. Pawson, Swansey Fold Farm, Whittle le Woods, Chorley, Lancs. LANCASHIRE.
July 16th, 1961. D. L. Chambers, Mustard Pot Hall, Eye, Suffolk. EAST SUFFOLK.
July 16th, 1961. L. Johnson, Elm Tree Farm, Moss Lane, Little Hoole, Preston, Lancs. LANCASHIRE.
July 16th, 1961. J. H. Wignall, Avondale, Dob Lane, Little Hoole, Preston, Lancs. LANCASHIRE.
July 17th, 1961. F. G. Hurren, Risings Farm, Bruisyard, Saxmundham, Suffolk. EAST SUFFOLK.
July 17th, 1961. Greetwell Poultry Products, Greetwell Hall, Manton, Gainsborough, Lincs. LINCOLN, PARTS OF LINDSEY.
July 17th, 1961. R. J. Aldrich, Rookery Farm, Laxfield, Woodbridge, Suffolk. EAST SUFFOLK.

Swine Fever

July 11th, 1961. J. Ellis, Werganrows Farm, Upper Killay, Bishopston, Swansea, Glam. GLAMORGAN.
July 11th, 1961. M. Hunt, Eastfield Farm, Halsham, Hull, Yorks. YORK, EAST RIDING.
July 11th, 1961. B. Whitlow, Railway View Farm, Nook Lane, Golborne, Warrington, Lancs. LANCASHIRE.
July 11th, 1961. J. A. Reynolds, Better View, Tostock, Bury St. Edmunds, Suffolk. WEST SUFFOLK.
July 11th, 1961. E. H. Willis, Lyppard Orchard, Trotshill, Worcester. WORCESTERSHIRE.
July 11th, 1961. The Officer in Charge, R.A.F. Station Farm, Benson, Oxford. OXFORDSHIRE.
July 11th, 1961. R. P. Stevenson, Blakemore House, Oulton, Norbury, Stafford. STAFFORDSHIRE.
July 12th, 1961. G. H. Whittaker, Shushions Manor Farm, Little Onn, Church Eaton, Stafford. STAFFORDSHIRE.
July 12th, 1961. H. Thomas, Nunnery House, West End, South Cave, Brough, Yorks. YORK, EAST RIDING.
July 13th, 1961. A. G. Skidmore, Fair View, Campion Lane, Hutton Rudby, Yarm, Yorks. YORK, NORTH RIDING.
July 13th, 1961. J. T. Snow, Fauld House Farm, Fauld, Burton-upon-Trent, Staffs. STAFFORDSHIRE.
July 13th, 1961. A. D. Latham, 20, Atherstone Street, Fazeley, Tamworth, Staffs. Disease at Fazeley Road Piggeries, Tamworth, Staffs. STAFFORDSHIRE.
July 13th, 1961. F. M. Walker, Holme House Farm, Carlton-in-Lindrick, Worksop, Notts. Disease at: Field Farm, Grove, Retford, Notts. NOTTINGHAMSHIRE.
July 14th, 1961. G. & J. Slater, The Lawns, Calverhall, Whitchurch, Salop. SHROPSHIRE.
July 14th, 1961. A. D. Derricutt, Yarlet Bank Farm, Yarlet, Stafford. STAFFORDSHIRE.
July 14th, 1961. L. M. Langley, Ham Western Farm, Shepton Mallet, Somerset. Disease at: Lower Ridgeway Farm, East Compton, Somerset. SOMERSET.
July 14th, 1961. W. K. Baker, The Willows, Scalby, Gilberdyke, Brough, Yorks. YORK, EAST RIDING.
July 17th, 1961. L. G. T. Simmonds, Court Farm, Court Lane, Burnham, Bucks. BUCKINGHAMSHIRE.
July 17th, 1961. T. Harrison, Chatt House Farm, Burton Pidsea, Hull, Yorks. YORK, EAST RIDING.
July 17th, 1961. W. J. Booker, Pandy Farm, Alltwen, Pontardawe, Swansea, Glam. GLAMORGAN.
July 17th, 1961. F. G. Redfern, Rossington Grange Farm, Rossington, Doncaster, Yorks. YORK, EAST RIDING.
July 17th, 1961. T. F. Thorne, Hillside, Tawstock, Barnstaple, Devon. DEVON.
July 17th, 1961. A. Orlikowski, Daniels Water Farm, Great Chart, Ashford, Kent. KENT.

HOLIDAY EXCHANGES

Now that the holiday period is upon us, members may be interested in the following requests for accommodation which we have received from the Continent.

France

Exchange accommodation is sought for the following children: Martine Chapeau (aged 7) from the Sarthe region; Philippe Marcillaus (aged 13) from Vienne; Jean-Piere Bonaiti (aged 15) from Doubs; and Michel Craplet (aged 15) from Seine.

Docteur-Veterinaire Barrenache of Saint Jean de Luz (Basses Pyrénees) would like to place his 18-year-old son with a veterinary family in England during the summer, and Madame Clement of Donzey, Nièvre, would also like to find accommodation for her son aged 15.

There are also a number of requests for accommodation as a paying guest: Mlle. Auvolat (aged 15) from the Savoie region; Catherine Roy (aged 16½) from Constantine; Marie-Claude Berger (aged 16) from Seine; and Rene Hermitte (aged 14) from Maroc who would like to come to England on an "*au pair*" basis.

Members of the profession who would like to take part in this holiday scheme are asked to contact Mrs. E. M. Stobo of 211, Yarborough Road, Lincoln.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-Mouth	Fowl Pest	Sheep Scab	Swine Fever
16th to 30th June, 1961		10	—	—	51	—	49
Corresponding period in	1960	8	—	—	22	—	60
	1959	5	—	—	18	—	48
	1958	4	—	—	8	—	103
1st January to 30th June, 1961		109	—	102	651	—	598
Corresponding period in	1960	128	—	14	1,321	—	599
	1959	127	—	9	417	—	601
	1958	91	3	111	309	—	691

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

SCIENTIFIC

Simultaneous Vaccination Against Distemper, Hepatitis and Leptospirosis

Sir,—As vaccination procedures are extended there is a natural desire to combine antigens when possible, so as to reduce the number of injections to be given at any one time. In recent years bivalent, trivalent and quadrivalant vaccines have been developed for use both in human and in veterinary medicine; more recently German workers have developed a quadrivalent vaccine for dogs.* These combined vaccines may be living or dead antigens or a mixture of both. Experience in the human and veterinary field has, however, shown that creating these polyvalent vaccines is not merely the simple task of combining monovalent agents. The size of dose injected has to be kept standard (usually at 1.0 ml. or 2.0 ml. for veterinary use), and adjustment in the relative amount of each antigen has to be made to prevent the more potent components masking the weaker.

For some time we have been developing a quadrivalent vaccine prepared from attenuated distemper virus and inactivated hepatitis and *Leptospira* antigens: it should serve simultaneously to immunise, in a course of 2 injections, against distemper, hepatitis, *Leptospira icterohaemorrhagiae* and *Leptospira canicola*. The first dose is given as a freeze-dried vaccine containing all 4 antigens; the second dose is given 2 weeks later and is a liquid vaccine containing hepatitis and *Leptospira* antigens only. During our work, groups of dogs from 4 to 20 in number, were employed in potency tests, in which studies were made of their ability to develop antibody and to withstand subsequent challenge. Supporting studies were also carried out on ferrets and hamsters. As was to be expected, some of the earlier material failed to produce circulating antibody or satisfactorily to protect against all 4 diseases, but whenever adequate levels of antibody were produced the experimental animals withstood direct challenge. Later it was found possible to produce a balanced antibody response by adjusting the proportions of the various antigens. Material of this kind is now available† and has been tested for antibody response on dogs, besides being submitted to the tests recommended in the British Veterinary Codex for distemper and *Leptospira* vaccines. The following table shows the response achieved by 6 dogs given a routine batch of the quadrivalent vaccine and tested for 14 days after the second inoculation.

Dog No.	Titre Neutralising Antibody		Titre Agglutinating Antibody	
	Distemper	Hepatitis	*L. ictero*	*L. canic.*
705	320	160	300	100
707	320	160	30	1,000
724	640	160	300	100
733	640	80	100	300
734	320	160	100	100
735	640	160	30	100

It will be seen that the values are similar to those found by Ablett, Uvarov, Addison and Baker (1959) after the simultaneous inoculation of the individual vaccines. A fuller report of this work will be submitted for publication in due course.

Yours faithfully,

S. A. KEEBLE,
KATHLEEN R. HEATH,
L. A. BAKER.

Glaxo Laboratories Ltd.,
Greenford,
Middlesex.

July 11th, 1961.

Reference

ABLETT, R. E., UVAROV, O., ADDISON, I. E., & BAKER, L. A. (1959). *Vet. Rec.* **71**. 774.

* Maxavac, Behringwerke.

† Canilep.

GENERAL

The Toxicity of Certain Ground Nut Meals to Poultry

Sir,—During 1960 it was estimated that 500 outbreaks of Turkey "X" disease occurred in this country involving the loss of 100,000 turkey poults (Blount, 1961). The feeding of Brazilian ground nut meal was found to be the cause of these outbreaks (Blount, *op. cit.*). A similar condition in ducklings which occurred in Kenya in 1960 (Haig, D. A., 1960, personal communication) was proved to be due to locally grown expeller-processed ground nut meal (Asplin & Carnaghan, 1961).

During the first 6 months of 1961 37 outbreaks of so-called "X" disease have been diagnosed at Weybridge. Twenty-eight of these occurred in turkeys between the age of 2 and 13 weeks; 8 involved ducklings and one outbreak was confirmed in pheasant chicks. Mortality in these outbreaks has not been as heavy as that experienced in this country during 1960. The material examined was obtained from birds sent to Weybridge for *post-mortem* examination and fixed specimens sent by Veterinary Investigation Centres for histological examination.

Diagnosis was based on the presence of histological liver lesions as described in turkeys by Siller and Ostler (1961) and in ducklings by Asplin and Carnaghan (1961). The lesions in pheasants have been found to be similar to those in turkeys.

The cases that have occurred this year have been in birds fed on proprietary rations produced by a number of firms. In those outbreaks in which we have been able to obtain details of the ration the compounders have stated that Indian ground nut meal was an ingredient.

Samples of two turkey rations which had been associated with outbreaks of "X" disease and which

contained approximately 6 per cent. of this ground nut meal were obtained. Each of these was fed to a group consisting of 6 day-old ducklings. Two other groups were fed a similar ration containing the same amount of Indian ground nut meal which was known to be non-toxic. (Day-old ducklings have been found to be highly susceptible to the toxic factor in ground nut meals (Asplin & Carnaghan, *op. cit.*).) After 12 days' feeding the average weight of the birds on the non-toxic rations was 10 oz. in both groups compared to 5.3 oz. and 5 oz. on the rations containing the suspected toxic ground nut meal. In each of these latter groups 5 of the ducklings died within 6 weeks of starting the experiment. The surviving bird in each group was killed on the 53rd day. The gross and microscopical liver lesions in all of these birds were identical to those produced by Brazilian and East African toxic ground nut meals. There were no losses in the control groups and the livers were normal on histological examination.

By comparison a ration containing 6.25 per cent. of toxic Brazilian ground nut killed all of a group of 6 day-old ducklings in 13 days.

Extracts of the Indian ground nut meal in these rations were prepared by developments of the method described by Allcroft *et al.* (1961). These were administered to 3 groups of 4 day-old ducklings so that each duckling received the equivalent to 100 grammes, 200 grammes and 750 grammes of ground nut meal in periods of 5, 5 and 11 days respectively. No deaths occurred in these birds but when they were killed 2 days after the termination of dosing, gross and microscopical liver lesions were present in all of them similar to those produced by feeding, and dosing with extracts of toxic Brazilian and East African samples. Control groups kept in the same brooder but not dosed were normal.

The information we have received indicated that the Indian ground nut meal had not been contaminated by contact with either Brazilian or East African meal in this country.

On the basis of these feeding and dosing experiments it appears that certain consignments of Indian ground nut meal contain a toxic principle similar to that found in Brazilian and East African samples in 1960. The toxicity of the Indian samples tested is considerably less than any of the toxic Brazilian or East African ground nut meals we have examined.

Yours faithfully,

R. B. A. CARNAGHAN.

Central Veterinary Laboratory,
Weybridge.

K. SARGEANT.

Tropical Products Institute,
London.

July 17th, 1961.

References

ALLCROFT, RUTH, CARNAGHAN, R. B. A., SARGEANT, K., & O'KELLY, J. (1961). *Vet. Rec.* **73.** 428.

ASPLIN, F. D., & CARNAGHAN, R. B. A. (1961). In press.

BLOUNT, W. P. (1961). *J. Brit. Turkey Fed.* **9.** 52.

SILLER, W. G., & OSTLER, D. C. (1961). *Vet. Rec.* **73.** 134.

Hypothermia in the New-born Puppy

Sir,—Observations similar to those recorded by Mr. Crighton (*Vet. Rec.* **73.** 646) have been noted at the Canine Health Centre. The article was therefore read with great interest and admiration for the volume of material that the author had collected.

Larin *et al.* (1958) reported that the average rectal temperature taken "immediately after whelping" was 74.9° F. in a normal Greyhound litter. Recent investigations into the physiological stability of the new-born puppy here have shown, in agreement with Mr. Crighton's findings, that such precipitate falls in post-natal temperature are normal in apparently healthy puppies. By recordings made every 10 minutes, it has been found that the temperature graph falls to 85° F. within the first 15 minutes of extra-uterine life, and that from then on to birth plus 60 minutes approximately, in the normal puppy, the temperature graph levels out and starts to rise but does not reach "normal" for some days.

This fall is apparently reduced but not eliminated by incubation at 97° F., and over a period of 160 minutes the rectal temperature does not tend to rise to that of the incubator.

Certain observations have indicated that a fall in rectal temperature of the puppy to as low as 67° F. in the early post-natal period is not incompatible with survival to at least 4 months of age.

It might appear, therefore, that hypothermia in the healthy puppy is not in itself lethal, nor is it an indication of the presence of a pathological process, unless the temperature continues to fall spontaneously after the normal period of initial hypothermia.

These observations in a seemingly neglected field of research are being continued, with various modifications and applied insults, in an attempt to throw some light on the causes of an abundance of unexplained neo-natal deaths in puppies. Would it be too presumptuous to suggest that this hypothermic phenomenon may be normal only in those mammals which at birth are largely incapable?

Yours faithfully,

J. M. HIME.

The Animal Health Trust,
Canine Health Centre,
Kennett,
Nr. Newmarket,
Suffolk.

July 10th, 1961.

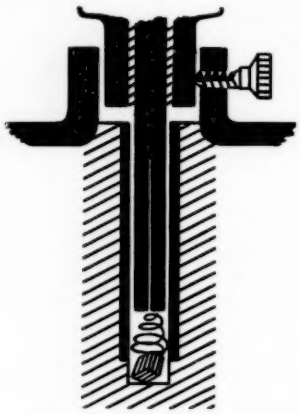
Reference

LARIN, N. M., SKULSKI, G., & ORBELI, W. G. (1958). *Brit. vet. J.* **114.** 112.

Another Modification of the Veterinary Auriscope

Sir,—I often have trouble with bad connexions between auriscope or ophthalmoscope heads and the battery holder. These are usually due to lack of springiness in the pins of the former.

I suggest the insertion of a small spring into the socket. This must be made of non-corrodible wire. (A suitable piece is provided in some thermometer cases.)



Some holders have an insulated base to the socket, so a piece of matchstick must be inserted first.

Yours faithfully,
DAVID UNWIN.

Taylors Cottage,
Chichester Road,
Midhurst,
Sussex.
June 25th, 1961.

Prolapse of the Uterus in the Cat

Sir,—Like Mr. Newman (*Vet. Rec.* **73**. 680) I have been presented recently with a "queen" suffering from uterine prolapse. In this case there were a few differences from that recorded by him and surgical intervention was also rather different.

The queen was a 6-year-old which had had numerous litters throughout her life, and always without any difficulty. On this occasion she gave birth to one live followed by one dead kitten, quite normally. According to the owner the uterus prolapsed almost immediately after the birth of the second kitten.

When examined some 2 hours or so later the cat appeared to be suffering no ill-effects at all from the prolapse, although, even in this short time, the uterus was grossly engorged and oedematous. Only the body and right cornu had prolapsed.

With the cat restrained, attempts were made to reduce the prolapse. These were not successful and as the mucosa of the uterus was very fragile, further attempts were abandoned and excision was decided upon.

The cat was anaesthetised with an ether/air mixture. It was thought impracticable to amputate in a manner similar to that described by Newman, because of the danger of rupturing the mesovarium of the left ovary. A midline laparotomy was performed and the left mesovarium was located, ligated and severed. The right ovary was located in the pelvis and traction at this stage failed to bring the ovary back into the abdomen. An incision was made into the ventral aspect of the prolapsed uterus beyond the cervix and the proximal part of the cornu was pushed back through the pelvis with the finger through this incision. This brought the right ovary into the abdomen and the mesovarium was similarly severed. After ligating the uterine vessels the circumferential incision in the uterus was completed and the whole of the organ was excised by drawing it through the pelvis. A purse-string suture was applied to the uterine stump and the stump was then replaced in the pelvis and transfixed to fascia at the pelvic brim. The laparotomy incision was closed in the usual manner.

Post-operative treatment consisted of 500,000 units of crystalline penicillin for 3 days. At no stage did the animal appear in any way distressed by the prolapse and the operation. Although the sutures are not due to be removed for another few days, the cat is remarkably well.

I am unable to think of any reason why the condition occurred in this queen. She was an active "hunting" cat in apparently a well-fed yet fit condition. The kittens although small in number were no larger than average, thus one assumes that no undue strain on the uterus was necessary for the birth to be completed. Perhaps the fact that the prolapse occurred shortly after the expulsion of the second kitten points to the reason in that, if placental separation were incomplete, some tension was made on the contracting uterus and this was sufficient to start the eversion which was completed by uterine force.

Yours faithfully,
J. LUCKHURST.

"Robwyn,"
Station Road,
Four Marks,
Alton,
Hants.
July 11th, 1961.

Re-vaccination of Guide Dogs for the Blind

Sir,—As a member of the Council of the Guide Dogs for the Blind Association, I have been asked to write a short note to you, on the subject of re-vaccination of guide dogs.

It is hoped that the cost of re-vaccination can be kept to a minimum for the blind persons owning guide dogs, and in consequence it has been arranged that vaccine will be distributed to the veterinary surgeons concerned, by the Guide Dogs for the Blind Association; the cost of the vaccine and its distribution to be borne by the Association.

It is presumed that such veterinary surgeons as have guide dogs in their practices, if approached, will be prepared to participate in these arrangements. The owners of guide dogs have been written to, and asked for the names of their veterinary surgeons, and as soon as a full list has been compiled, a letter will be sent to each veterinary surgeon asking for his or her co-operation.

Yours faithfully,
S. F. J. HODGMAN.

The Animal Health Trust,
Canine Health Centre,
Kennett,
Nr. Newmarket,
Suffolk.
July 12th, 1961.